Imaging of Traumatic Arterial Injuries to the Cervical Vessels

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Trauma involving the cervical region can result in either blunt or penetrating injury to the cervicocerebral vessels, with attendant hemorrhagic and/or neurologic sequelae. The reported incidence of carotid or vertebral artery injury in all trauma patients is 1.2–1.6 % [1], with an associated risk of acute cerebral ischemia in 12–15 % of affected individuals. Blunt cervical vascular injury (BCVI) is increased in the setting of cervical spine, basilar skull, or severe facial fractures; spinal cord and traumatic brain injury; major thoracic injuries; and cervical hyperextension/rotation or hyperflexion [2]. The vertebral artery is more commonly injured than the carotid artery because of its close proximity to bone as it runs through the intervertebral foramina. Twenty percent of BCVI patients, however, demonstrate none of these "classic" risk factors. Damage from penetrating cervical vascular injury (PCVI) trauma is less common than blunt trauma, with carotid and vertebral artery injuries accounting for only 3 % and 0.5 %, respectively, of arterial injuries in civilians.

Anatomical Considerations

Specific anatomical features are important in the evaluation of blunt trauma. The internal carotid artery (ICA) courses ventral to the transverse processes from C1 to C3 before it enters the petrous canal at the skull base. Injury from hyperextension and contralateral rotation occurs when the vessel impinges upon the lateral articular processes and pedicles of the upper cervical spine. The ICA is also vulnerable to dissection at the skull base from deceleration injury or petrous canal fractures. The vertebral artery's course through the C2–C6 transverse foramina predisposes it to injury from subluxation or rotation or from transverse process fractures. The distal cervical (V4) segment of the vertebral artery may be crushed against the C1 vertebra or the dural edge in cases of craniocervical junction distraction or dislocation.

Historically, the approach to diagnosis and treatment of PCVI starts by determining the location of the injury within one of three anatomic zones anterior to the sternocleidomastoid muscles. The diameter of all three vascular structures (vertebral artery, carotid artery, and internal jugular vein) is greater, and their location more superficial, as the anatomical plane moves caudally. Zone I contains the origins of the brachiocephalic vessels and the subclavian and innominate veins. Here, vascular structures are most vulnerable to small fragments and shallow wounds, and penetrating injuries in this location carry the highest morbidity and mortality. Zone II (Fig. 1a) includes the distal common carotid arteries, the proximal internal and external carotid arteries, the vertebral arteries, and the internal jugular veins. The vertebral artery remains narrow and the furthest from the skin surface and is protected by 4–6 mm of bone throughout its course except in Zone I. Zone III (Fig. 1b) holds the distal cervical internal carotid arteries, the external carotid artery branches, the distal vertebral arteries, and the proximal internal jugular veins. The carotid artery and internal jugular vein are less vulnerable in Zone III due to protection from the mandible and smaller size of the vessels; the zone's posterior portion is the least common area of the neck to be injured by penetrating trauma.

Penetrating injuries in Zone II with life-threatening hemorrhage, expanding hematoma, airway compromise, or loss of the carotid pulse with a neurological deficit are often explored surgically, although patients with stable vital signs are often evaluated first by noninvasive imaging. For example, all three zones can be rapidly assessed using computed tomographic angiography (CTA), and multiplanar reconstruction in bone windows detects vertebral, skull base, and/or facial fractures associated with vascular injury [4].

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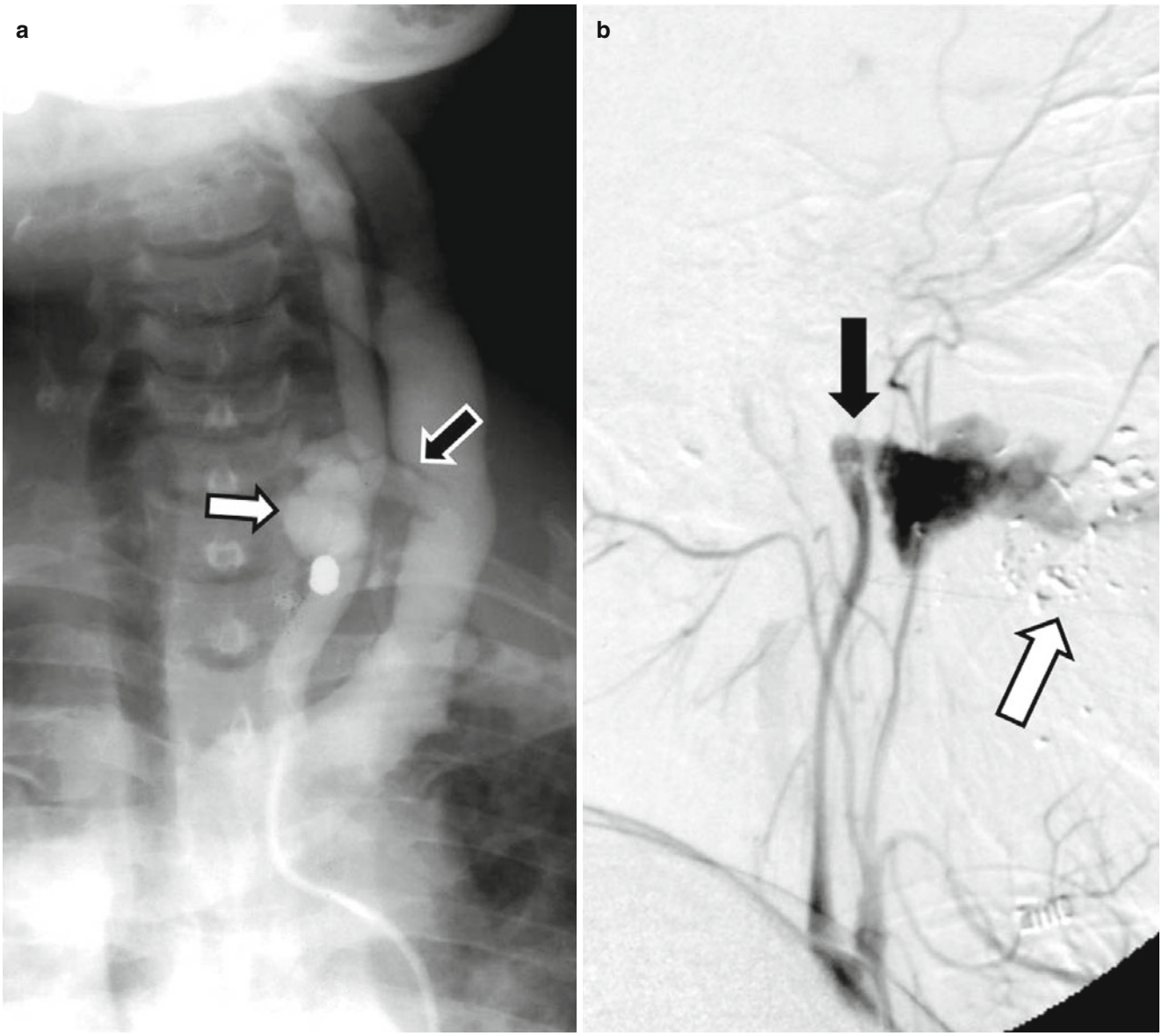


Fig. 1 (**a**, **b**) Penetrating injuries. (**a**) Zone II. AP view of a catheter angiogram (CA) done on a gunshot victim. A bullet fragment overlies the left common carotid artery (CCA) with an adjacent pseudoaneurysm (*white arrow*) and a CCA to internal jugular vein arteriovenous fistula (*black arrow*). (**b**) Zone III. Lateral digital subtraction angiogram (DSA) view of a self-inflicted gunshot to the mouth shows transection (*black arrow*) of the distal left cervical ICA with extravasation along the bullet path. Multiple bullet fragments are noted in the soft tissues (*white arrow*)

Imaging Modalities

Medical imaging is a central component of the diagnostic evaluation of vascular injuries. Each imaging modality plays a unique role in the diagnosis of these lesions.

Duplex Sonography

Duplex sonography is ideal for use in the emergency department as it is portable and requires no contrast. Doppler imaging demonstrates endoluminal flow characteristics, while gray-scale imaging provides detailed imaging of the vessel wall and extraluminal structures. Occlusion, dissection, intramural hematoma, pseudoaneurysm, laceration, transection, or arteriovenous fistula have been reliably identified in several studies [8, 9], with a reported sensitivity as high as 92–100 % in penetrating neck trauma.

Ultrasound has several limitations in the evaluation of traumatic lesions. The study is time-consuming and operator dependent; metallic foreign bodies, subcutaneous gas, and osseous structures limit visibility of the vascular structures;

and detailed evaluation is particularly difficult for vessels located above the angle of the mandible or running through bony foramina.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) offers a high degree of tissue contrast and spatial resolution that allow for reliable disease detection. In the setting of cervical trauma, MRI is the test of choice to evaluate the spinal cord, brachial plexus, and musculotendinous and ligamentous elements. MRI also provides the most effective evaluation of the intracranial compartment for ischemic and hemorrhagic complications of cervical vascular injury.

Two- and three-dimensional time-of-flight (TOF) and contrast-enhanced MR angiography (MRA), as well as fat-saturated T1- and T2-weighted sequences, are effective at evaluating arterial wall integrity, especially when arterial dissection is suspected [10].

In the largest series of MRI use in suspected dissections, Levy et al. reported an overall sensitivity and specificity of 83 % and 99 %, respectively, using 3D-TOF angiography, although sensitivity was much lower for the vertebral arteries (20 %).

MR imaging of cervical vascular injury is limited by the presence of metallic fragments, the lack of detailed evaluation of the osseous structures, and time constraints associated with seriously injured individuals. In the United States, emergency department imaging of cervical trauma relies primarily on computed tomography (CT).

Computed Tomography

With the development of multi-detector CT (MDCT) technology, computed tomography is the predominant triage tool for trauma patients [11]. High spatial resolution and isometric voxelation allow for seamless multiplanar reconstructions and volume-rendered three-dimensional imaging of large tissue volumes. In cervical trauma, MDCT can image the entire cervicocerebral vascular system within a few seconds, along with the adjacent soft tissue and osseous structures. CT imaging carries no absolute contraindications and very few relative contraindications, e.g., contrast allergy, which allows for safe and rapid performance of the procedure.

Computed tomographic angiography (CTA) has a spatial resolution that rivals catheter angiography. A recent evaluation of CTA with 16-slice MDCT scanners demonstrated 97.7 % sensitivity and 100 % specificity compared to conventional angiography. Because of these attributes, MDCTA has usurped catheter angiography as the predominant imaging modality in the diagnosis of cervical vascular injury [7].

Digital Subtraction Angiography

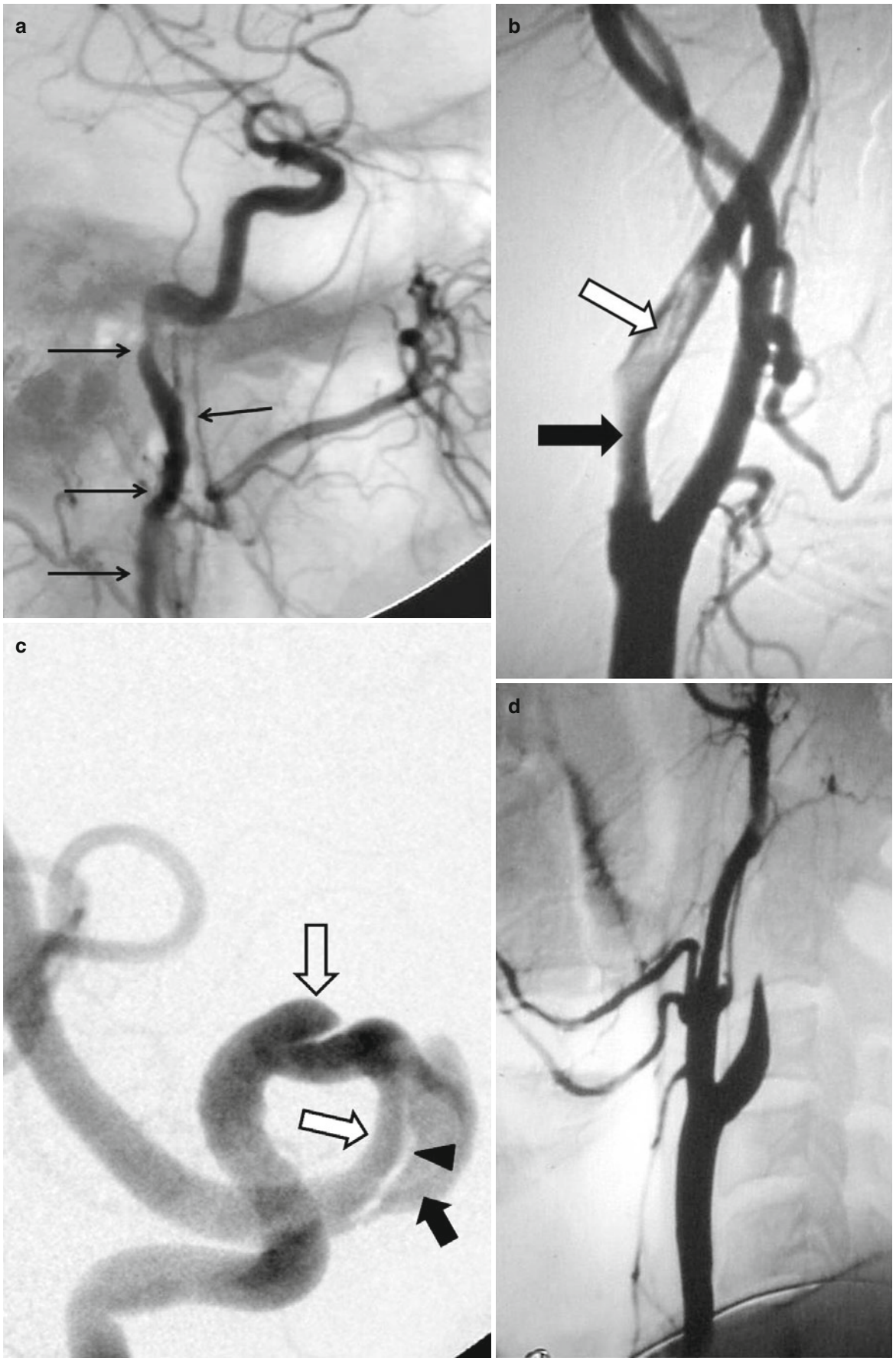
For years, digital subtraction angiography [DSA] represented the "gold standard" for imaging evaluation of vascular injuries. However, continued improvements in noninvasive vascular imaging have caused a shift away from this invasive technology for the bulk of the diagnostic evaluation. Two major limitations in DSA contribute to this shifting diagnostic paradigm—DSA is limited in its assessment of the extravascular structures and mural integrity, and there is the potential for neurologic and non-neurologic complications. On the positive side, DSA allows for very high spatial resolution, as well as provides temporal information relating to the hemodynamics of the cerebrovascular tree. DSA remains the test of choice for detecting flow-related complications of cervical and cerebral vascular injury, such as arteriovenous fistulas, and for the evaluation of collateral circulation. DSA is often used when noninvasive imaging is inconclusive or necessary for preoperative planning and as an endovascular alternative to open surgical vascular repair.

Patterns of Injury

Intimal damage is the final common pathway for vascular injury, regardless of the mechanism of action. Even minimal intimal disruption may promote the cascade of platelet aggregation and clot formation, leading to distal embolization or vascular thrombosis.

Focal spasm or mild luminal irregularity (Fig. 2a) may be all that is noted. However, more substantial injury includes subintimal dissection with (Fig. 2b) or without intramural thrombus, raised intimal flap (Fig. 2c), pseudoaneurysm formation (Figs. 1a and 2c), occlusion (Fig. 2d), transection with active extravasation (Fig. 1b), and arteriovenous fistula (AVF) development (Fig. 1a). Progression of subintimal thrombus in a false lumen or a subendothelial tear with false channel (Fig. 2c) or pseudoaneurysm enlargement may lead to luminal stenosis with subsequent hemodynamic compromise and cerebral ischemia. Rapid change in the appearance of the injury can occur. Combinations of injuries may occur in the same vessel (Fig. 1a), and multivessel injury has been reported in 18–38 % of cases.

In an effort to predict the risk of stroke, Biffl et al [5]. devised a grading scale based upon the angiographic appearance of blunt carotid injuries. The scale was modified in 2002 to include arteriovenous fistulas (AVFs) and to apply also to vertebral artery injuries. Grade I lesions are vessels which show luminal irregularity with less than 25 % luminal narrowing (Fig. 2a). Grade II lesions are those with dissection or intramural hematoma with greater than 25 % luminal narrowing (Fig. 2b), intraluminal thrombus (Fig. 2b), raised intimal flap (Fig. 2c), or hemodynamically insignificant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arteriovenous fistula (AVF). Grade III lesions are pseudoaneurysms (Figs. 1A and 2c); Grade IV, vessel occlusion (Fig. 2d); and Grade V, transaction (Fig. 1b) or hemodynamically significant AVF (Fig. 1a). In the carotid territory, the higher the Biffl grade, the higher the risk of stroke. In the vertebral system, Grade II lesions carry the highest risk.

It is important to recognize that vascular injury, usually in the form of a dissection, can occur after trivial trauma or in otherwise healthy individuals with no obvious risk factors. The average annual incidence of these "spontaneous" occurrences is between 2.6 and 2.9 per 100,000 people and accounts for 13–22 % of ischemic strokes in patients younger than 45 years of age. Known collagen-vascular disorders (CVDs), such as Ehlers-Danlos syndrome (Type IV), fibromuscular dysplasia, and Marfan's syndrome, are associated with the development of spontaneous dissection. Skin biopsies in patients with spontaneous dissection often demonstrate structural abnormalities of their connective tissue, making them more prone to injury from insignificant trauma such as coughing or sneezing [3].

Vasospasm

Vasospasm is a physiological response of the arterial wall to mechanical or chemical irritation resulting in contraction of the smooth muscle within the wall and appearing as segmental areas of associated vascular narrowing on imaging. Vasospasm is a uniformly reversible and self-limited event and usually responds favorably to vasodilators such as nitroglycerine, papaverine, and calcium channel blockers.

Vasospasm can mimic subtle intimal injury, which can manifest as segmental narrowing, but vasospasm should never be associated with an intimal flap or pseudoaneurysm. It can also be indistinguishable from mild forms of connective tissue disorders, particularly fibromuscular dysplasia, and can be subtle enough to be overlooked or undetectable on CTA and MRA.

Intimal Flap

An intimal flap is the separation of a short segment of the intimal layer from the medial layer of the arterial wall. It appears on imaging as a linear intraluminal filling defect that is in continuity with the arterial wall, but without an associated wall hematoma or distinct false lumen (Fig. 3a). Intimal flaps have similar imaging characteristics on CTA and MRA, but they are more reliably detected with CTA (Fig. 3b). Because they are focal and often subtle, the ability to detect them on imaging is impaired by adjacent metallic foreign bodies or other sources of beam-hardening artifact, such as venous contrast reflux into the internal jugular vein, or by quantum mottle caused by the shoulders.

Intimal flaps can be a nidus for, and a mimic of, thromboembolus formation. They may also progress to frank dissection. In practice, the vast majority of minor intimal injuries heal spontaneously without sequelae. There is considerable debate over the incidence, significance, and appropriate treatment of intimal flaps. Observation may be all that is necessary, but when appropriate, short-term use of an antiplatelet agent, e.g., aspirin, can be considered.

Dissection and Pseudoaneurysm

Cervical arterial dissections are the most common vascular injury following blunt cervical trauma, representing as many as 76 % of these injuries, but they can also occur after mild trauma, i.e., chiropractic manipulation, roller coaster riding, or spontaneously without an identifiable traumatic event. There is clearly an association between spontaneous dissections and hereditary connective tissue disorders, although the estimates of the prevalence in the spontaneous dissection population vary dramatically, ranging from 0 to 18 %. Cervical arterial dissections can also arise as an extension of aortic aneurysms involving the aortic arch.

Pathophysiologically, arterial dissection represents a separation of the intimal layer of the arterial wall away from the medial layer, with the creation of a false channel between the two layers (Fig. 2c). Radiographically, cervical arterial dissections demonstrate a variety of appearances ranging from an uncomplicated intimal flap to complete occlusion. A dissection may look like a blind-ended pouch with an adjacent stenotic lumen, often eccentric, and varying degrees of contrast opacification (Fig. 4). If blood in the false channel creates a "reentrance" intimal tear or fenestration, the vessel takes on the "double-barrel" appearance of two parallel vascular channels (Fig. 2c), which, upon healing, may become permanent (Fig. 5). If the hemorrhage does not create a fen-

Fig. 2 (**a**) Left CCA lateral DSA view of a patient in a motor vehicle accident (MVA) with neck pain. The study shows mild luminal irregularity along the course of the distal ICA (*black arrows*) to the carotid canal indicative of intimal injury (Biffl Grade I). (**b**) Right CCA lateral DSA view of another MVA patient with stroke shows luminal narrowing of the carotid bulb of greater than 25 % (*black arrow*) consistent with an intraluminal hematoma and adherent intraluminal thrombus (*white arrow*) (Biffl Grade II). (**c**) Left vertebral artery (VA) oblique submentovertex DSA view of a patient in a snowboarding accident. A large intimal flap (*arrowhead*) is identified with compression of the true lumen (*white arrow*) by the false lumen (*black arrow*) (Biffl Grade II). In addition, a pseudoaneurysm is identified proximal to the flap (*open arrow*) (Biffl Grade III). (**d**) Right CCA lateral DSA view of an MVA patient with stroke shows complete occlusion of the internal carotid artery (Biffl Grade IV)

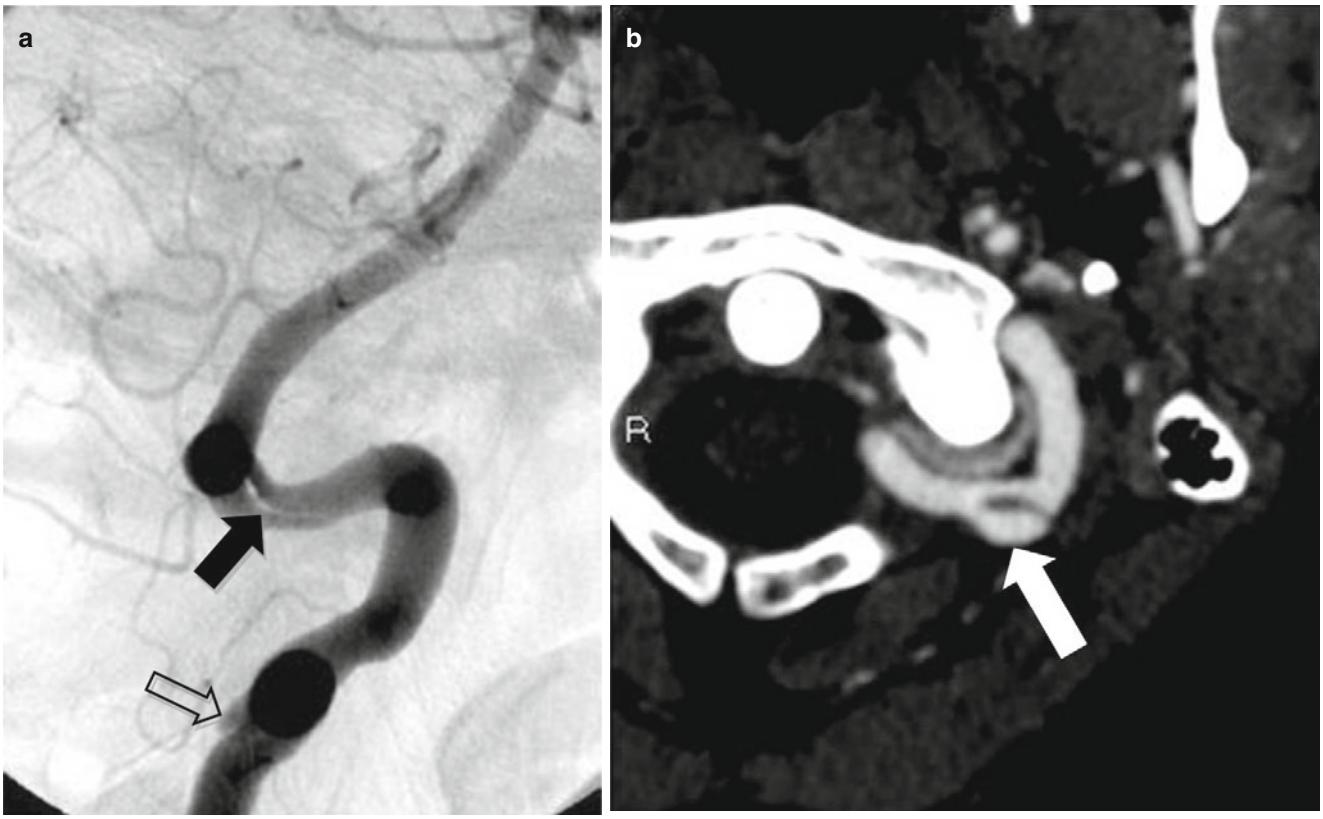

Fig. 3 (**a**) Left VA lateral DSA view in a patient with an athletic injury. A small intimal flap is identified (*black arrow*) in addition to a small intimal irregularity seen more proximally (*open arrow*). (**b**) A subsequently CTA shows the flap well on the axial view, with some early pseudoaneurysm formation (*white arrow*)

estration, the false lumen may lead to stenosis or occlusion of the true lumen or pseudoaneurysm formation. When the dissection involves the *subintimal* media, it will evolve into stenosis (Fig. 4) or occlusion (Fig. 2d). Stenosis usually appears as a smooth, tapered narrowing that varies in severity and length depending on the extent of the dissection. DSA imaging can elucidate the luminal narrowing associated with dissection, but cross-sectional imaging optimizes visibility of the offending intramural hematoma. On CTA, this manifests as luminal narrowing with paradoxical enlargement of the total diameter of the vessel (Fig. 6a). Intramural hematoma may appear iso- or mildly hyperdense to adjacent muscle. On MRI, the intramural hematoma becomes even more conspicuous on fat-saturated T1-weighted images, appearing as an intramural hyperintensity paralleling the vascular flow artifact (Figs. 6b and 7b) or as a hyperintense "crescent" sign on images perpendicular to the long axis of the vessel (Fig. 7a). The intramural thrombus can also be seen on susceptibility-weighted imaging (SWI) as a hypointense area (Fig. 6c) and will restrict diffusion on diffusion-weighted imaging (DWI). These findings are more reliably conspicuous in the carotid artery (Fig. 7) than the vertebral artery, as normal sluggish flow in the closely approximated vertebral venous system can mimic the "crescent" sign of vertebral dissection.

Occlusive dissection possesses a characteristic flame-shaped, tapered luminal narrowing (Figs. 2d and 8). The wall hematoma will possess the same imaging characteristics as described above, but differentiating intramural hematoma from intraluminal thrombus can be difficult, if not impossible, in the setting of occlusion.

Pseudoaneurysms (Figs. 1a, 2c, and 4) result from *subadventitial* dissection and appear as a saccular outpouching projecting beyond the expected confines of the vessel wall. The term "pseudoaneurysm" is used due to the fact that the aneurysmal outpouching does not consist of all three vascular wall layers. On DSA and cross-sectional vascular imaging, pseudoaneurysms appear as contrast-filled outpouchings adjacent to and in continuity with the vessel lumen (Fig. 4). On cross-sectional imaging, adjacent hematoma may be evident. Color-flow Doppler shows the "yin-yang" appearance of swirling flow within the pseudoaneurysm sac.

Pseudoaneurysms may form from a dissection caused by blunt injury or by penetrating trauma. Unlike dissecting pseudoaneurysms, pseudoaneurysms caused by laceration contain no vessel wall (Fig. 9), with the sac instead being

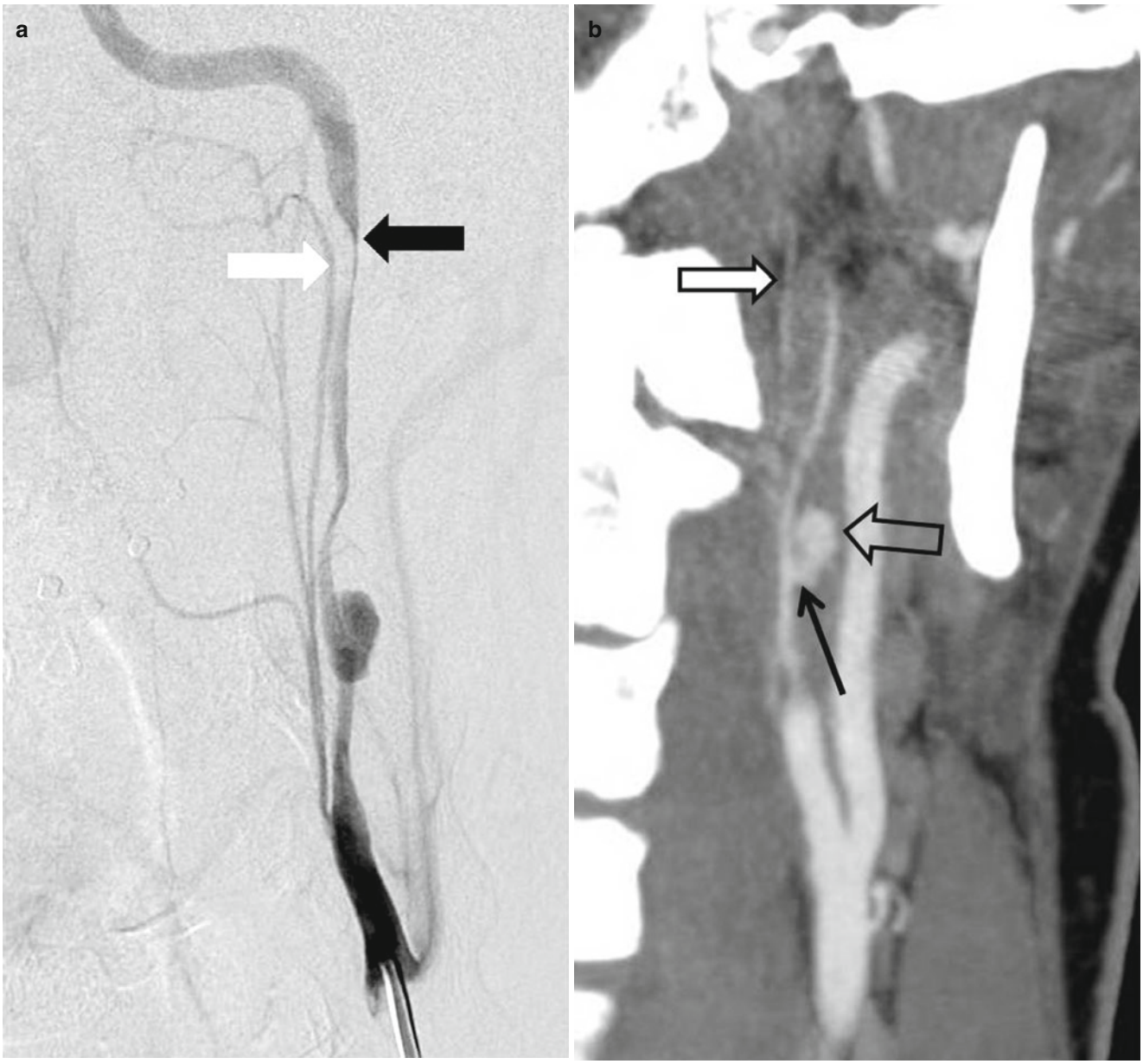

Fig. 4 (**a**) Left CCA DSA, AP view shows a subacute dissection from an MVA. There is abrupt tapering of the internal carotid artery from the C3 level to the carotid canal, where the lumen returns to normal diameter (*black arrow*). A blind-ended pouch representing contrast in the proximal portion of the dissection with pseudoaneurysm formation is noted. (**b**) The corresponding CTA shows the luminal narrowing and the false lumen pouch (*black arrow*), which extends outside of the vessel lumen as a pseudoaneurysm (*open arrow*). The ascending pharyngeal artery (APA) (**a,b** *white arrows*) parallels the ICA lumen and may be mistaken for a "string sign" in patients who actually have ICA occlusion. The APA, however, does not enter the carotid canal

comprised of perivascular connective tissue and/or surrounding hematoma. This results in an instability that invariably leads to pseudoaneurysm growth and could lead to a vascular "blowout." For these reasons, pseudoaneurysms related to vessel laceration are usually treated surgically or endovascularly.

Follow-up imaging in cervical arterial dissections is important, as dissections show a variable evolution. Many dissections heal without obvious abnormality, and follow-up imaging will appear normal or with minimal luminal irregularity. However, some dissections heal with chronic stenosis or pseudoaneurysm. Occlusive dissections may recanalize and become a source for distal embolization; dissecting pseudoaneurysms may enlarge and compress the adjacent vessel (Fig. 10). Understanding these changes aids in treatment decisions both in the acute and chronic stages of the disease.

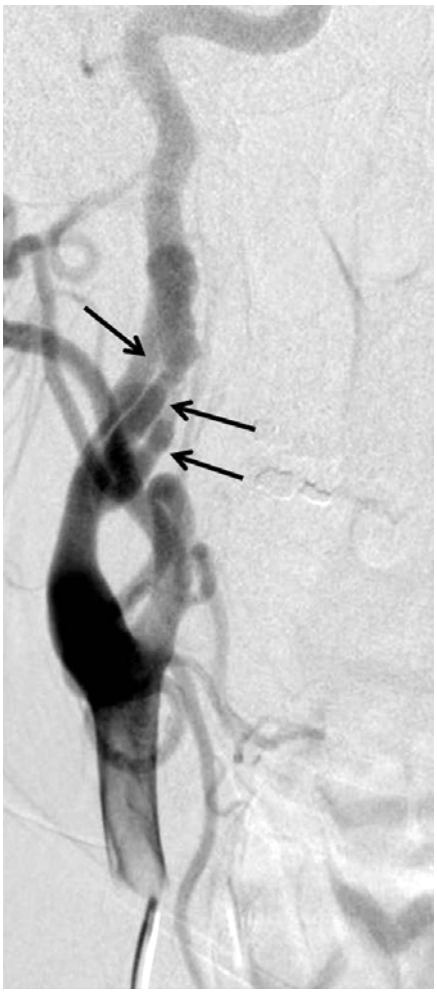

Fig. 5 Right CCA DSA, AP view in a trauma patient shows evidence of a healed dissected ICA with three separate channels (*arrows*)

Occlusion

Vascular occlusion is the most common imaging appearance of cervical carotid arterial injury in both blunt and penetrating cervical trauma, with a prevalence as high as 33 % and 36 %, respectively. The imaging appearance of occlusion varies relative to the acuity and etiology of the event. In situ thrombosis often demonstrates an abrupt, blunt occlusion that ends at or near a branch point (Fig. 11). Contrast tracking around intraluminal thrombus may appear as a "meniscus" or "tram-track" sign, and vessels tend to thrombose retrograde to a branch point. Conversely, occlusive dissection may be seen as a tapered, flame-shaped occlusion (Fig. 1d).

In CTA, arterial occlusion is usually readily apparent as an abrupt termination of the contrast column. Occlusion of a vessel in the early arterial phase may show no contrast enhancement within its lumen, misidentifying the level of the lesion. However, contrast opacification to the level of the occlusion occurs eventually as contrast material slowly percolates into the stagnant column and will be identified on delayed images if obtained.

In the acute phase of cervical arterial occlusion, collateral channels distal to obstruction may reconstitute the vessel downstream of the obstruction. Patterns of collateralization vary depending on the vessel and the site of the occlusion. In common carotid artery occlusion, retrograde filling of the external carotid artery to the internal carotid artery is often seen. In cervical ICA occlusion, antegrade filling of the intracranial ICA can occur through ECA collaterals with the petrous and cavernous segments of the ICA and via retrograde flow from the ophthalmic arteries. In cervical vertebral artery occlusion, potential collateral pathways at each vertebral level exist for vertebral artery reconstitution. Robust collaterals from the thyrocervical and costocervical trunks, as well as muscular vertebral and occipital artery branches, often revascularize an injured vertebral artery just distal to the occlusion.

When satisfactory collateral circulation is not present to maintain distal flow, the artery will thrombose retrograde to the last point of inflow or may consist of several discontinuous segments of filling. Differentiating between occlusion and severe stenosis is important because of the potential for future thromboembolic events in vessels with slow flow. As a noninvasive study, CTA shows a high degree of accuracy for differentiating these two entities, but when the results are inconclusive, catheter angiography can be performed to delineate between the two with a high degree of certainty.

Contrast-enhanced MRA (CE-MRA) results in similar imaging characteristics to those described above for CTA although its lower spatial resolution makes it more difficult to differentiate between occlusion and severe stenosis. Time-of-flight (2D and 3D) MR angiographic techniques have even less sensitivity and specificity than contrast-enhanced MRA.

Arteriovenous Fistulas

Arteriovenous fistulas (AVFs) are abnormal communication between arteries and veins that result in arterialization of the venous systems. Traumatic cervical AVFs almost always arise from penetrating trauma (Fig. 1b), although blunt trauma is implicated in some cavernous-carotid and vertebrovenous fistulas. Although not always the case, AVFs may be unsuspected initially, but enlarge and mature over time with symptoms presenting in a delayed fashion. Audible bruit or neck pain may indicate the presence of a fistula, but the initial signs may be more ominous, such as those related to cerebral ischemia, venous hypertension, or high-output failure.

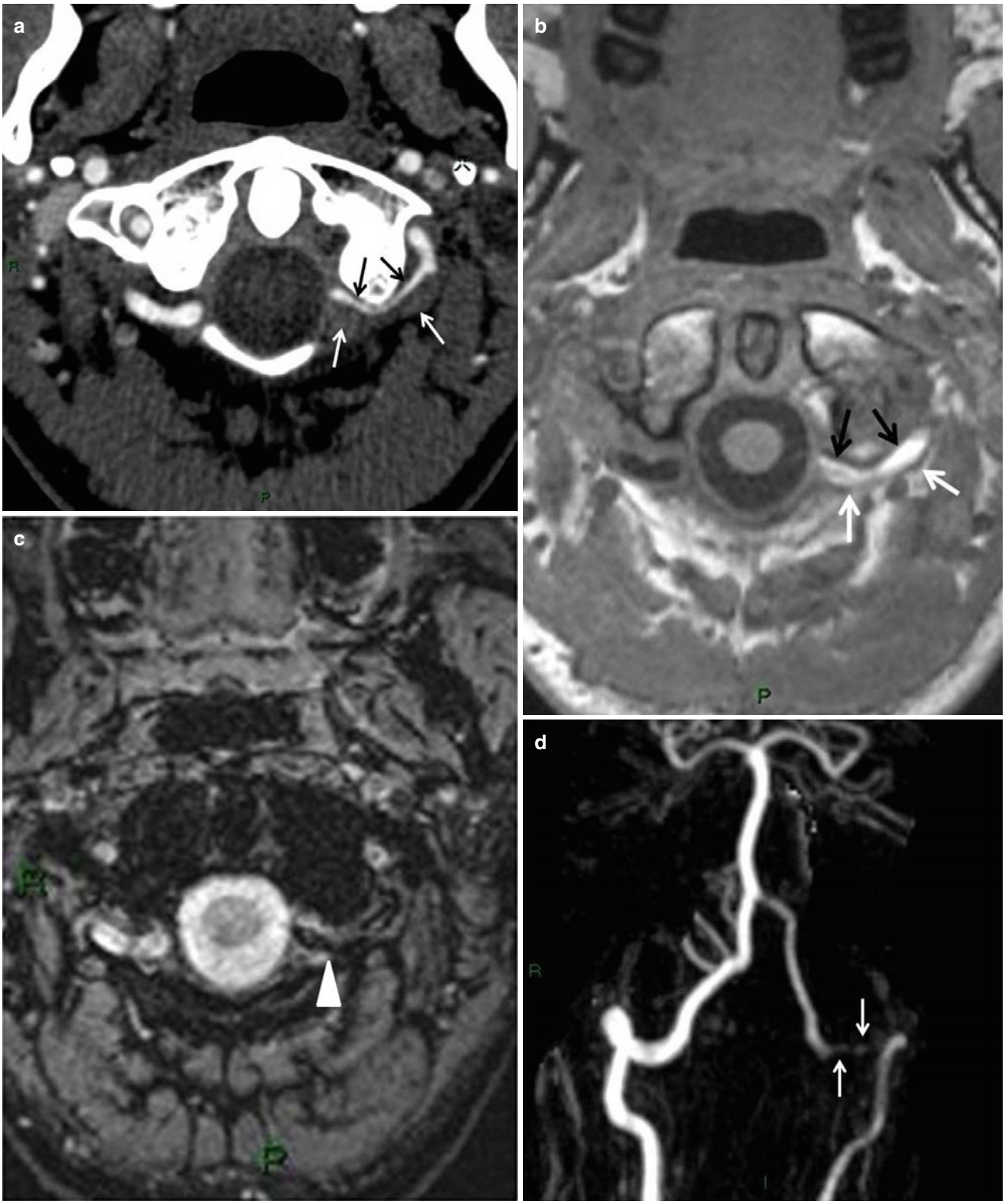

Fig. 6 (**a**) Axial CTA image through the distal vertebral arteries in a patient in a minor car accident with neck pain. The study shows compression of the contrast-filled lumen (*black arrows*) by intramural thrombus in the dissected segment (*white arrows*). Notice how the overall diameter of the vessel (distance from the *black arrows* to the *white arrows*) is larger than the diameter of the normal right vertebral artery. (**b**) Corresponding axial T1-weighted MRI shows the flow void within the compressed lumen (*black arrows*) and the hyperintense mural thrombus (*white arrows*). (**c**) The intramural thrombus on susceptibility-weighted imaging (*arrowhead*) is hypointense, and the contrast-enhanced MRA (**d**) shows the irregular and narrowed residual lumen through the affected area

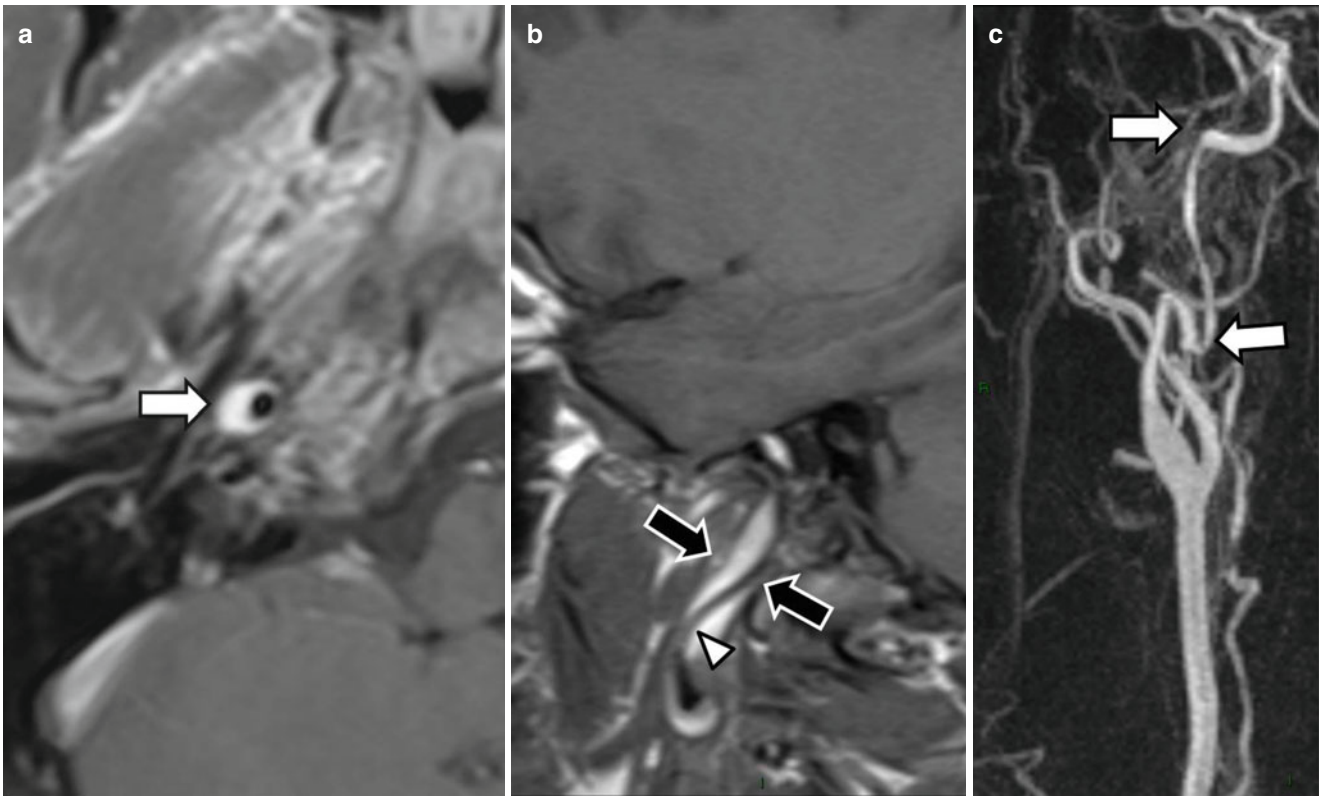

Fig. 7 MRI and MRA in a patient who sustained a fall show the "crescent sign" (*white arrow*) involving the right ICA at the skull base on axial T1-weighted imaging (**a**). The sagittal view nicely demonstrates the enlarged vascular structure with intramural thrombus (*black arrows*), and the residual lumen flow void coursing through the center (*arrowhead*). The corresponding gadolinium-enhanced MRA (**c**) shows the rapid tapering of the distal ICA with variable lumen size from the cervical loop to the vertical portion of the petrous carotid segment (*white arrows*)

Noninvasive vascular imaging may be able to detect obvious AVFs, but catheter angiography remains the gold standard or evaluating these lesions. Cross-sectional imaging may show a caliber change of the affected artery and enlargement of the affected vein. In some cases the fistulous connection is identified, particularly with time-resolved CT and MR sequences; detection and characterization of these lesions noninvasively remain suboptimal. If the lesion is in a location that can be studied with color-flow and spectral Doppler, ultrasound may show low-resistance arterial and arterialized venous waveforms or the actual fistulous connection. But these lesions are obvious on DSA, demonstrating the enlarged feeding artery, rapid arteriovenous transit time, and early filling of the affected veins. The size and location of the fistulous connection are identified, and the presence of collateral circulation can be identified in cases of arterial steal. In some cases, the fistula can be closed by endovascular means at the same time as the diagnosis (Fig. 12).

Transection

Transection represents the most severe form of cervical arterial injury and is often lethal. Penetrating trauma is the most likely cause of vascular transection, but excessive blunt force can be the inciting factor, particularly in severe spinal and skull base fractures. Radiographically, arterial transections can present with any of three imaging appearances: vessel occlusion (Fig. 9), active extravasation (Fig. 1b), or arteriovenous fistula (all described above). High-flow and/or retrograde steal may give the appearance of a transection in the presence of an arteriovenous fistula; however, antegrade flow is restored after fistula closure, demonstrating vessel patency.

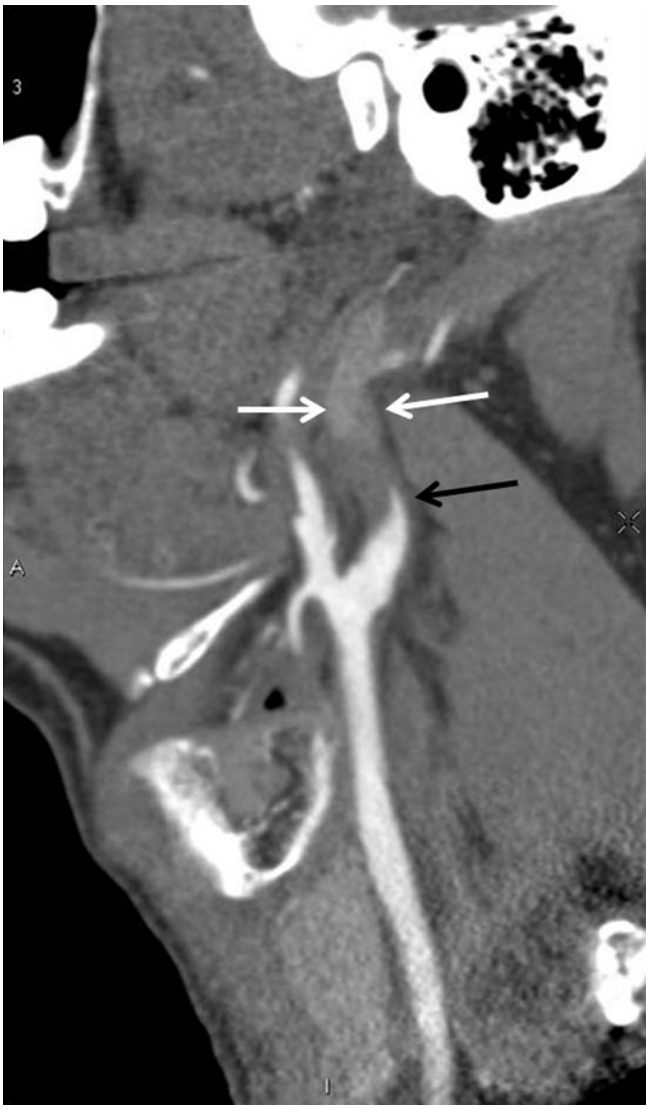

Fig. 8 Sagittal CTA in a patient with spontaneous carotid dissection shows the flame-shaped, rapid tapering of the ICA, which is occluded (*black arrow*). The enlarged, thrombus-filled distal carotid artery is seen (*white arrows*) and consistent with a thrombosed dissection. Intramural thrombus cannot be distinguished from intraluminal thrombus

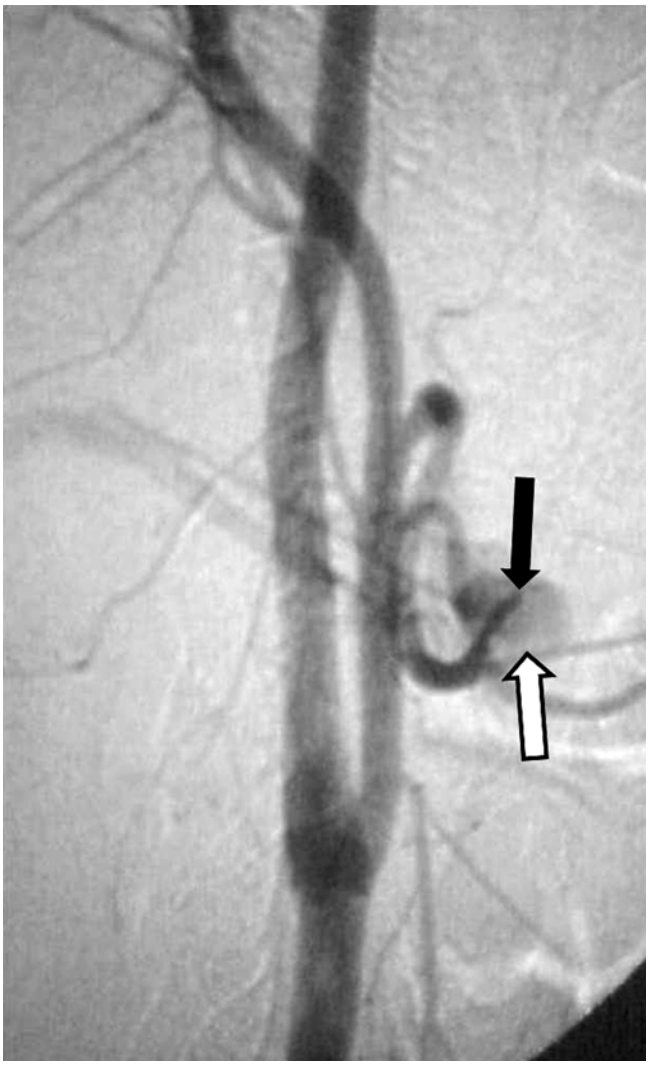

Fig. 9 Left CCA DSA, lateral view, in a farmworker who fell onto his shears, with a resulting injury to the facial artery. The vessel is transected (*white arrow*), and a traumatic pseudoaneurysm is identified at the site of the injury (*black arrow*). The vessel was completely occluded endovascularly using platinum microcoils (not shown)

Treatment

Image-guided therapy has evolved into the first line of treatment for many traumatic cerebrovascular injuries [6]. When DSA is employed in the diagnosis of these lesions, endovascular techniques and appropriate devices allow treatment to take place at the same time. Flow-limiting or symptomatic dissections can be treated with balloon angioplasty or endovascular stenting; pseudoaneurysms are now being closed with coil embolization or the placement of a flow diverter. Life-threatening transections and active extravasation can be treated with vessel sacrifice; and arteriovenous fistulas can be closed via transarterial (Fig. 12) or transvenous embolization. Unfortunately, a detailed discussion of these exciting techniques is beyond the scope of this syllabus.

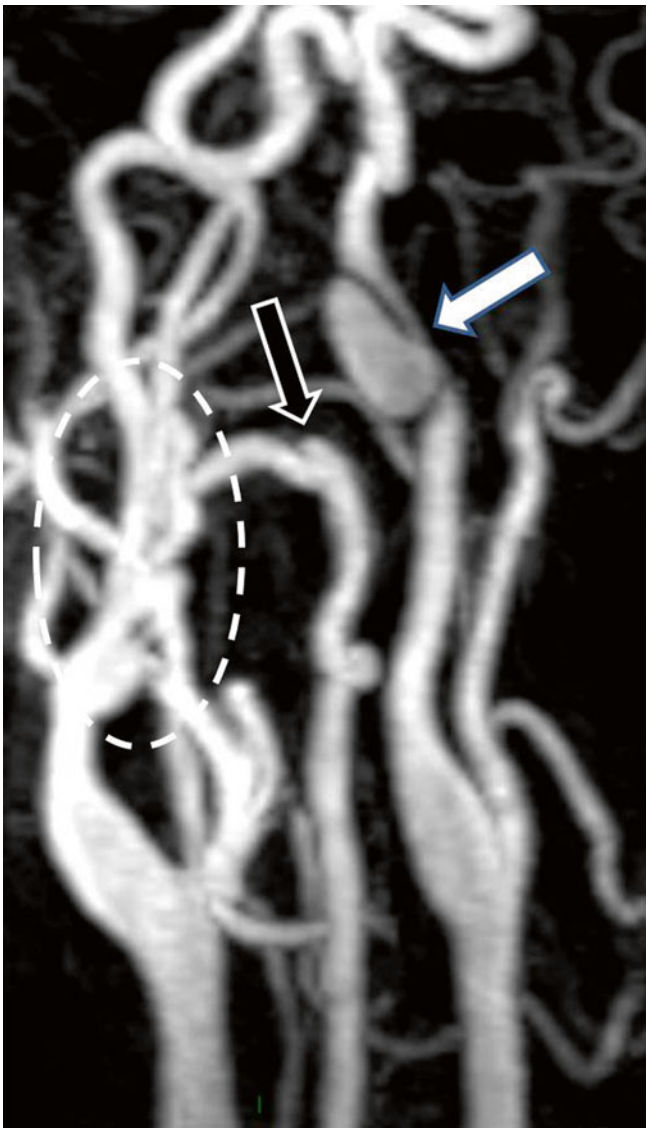

Fig. 10 Follow-up contrast-enhanced MRA of patient with multiple vascular injuries and of the same patient as in Fig. 5. This study shows a stable multichannel right ICA dissection (*dashed circle*); a small, stable left vertebral artery pseudoaneurysm (*black arrow*); and a progressively enlarging left ICA pseudoaneurysm causing significant stenosis of the left ICA (*white arrow*). The pseudoaneurysm was treated with carotid stenting (not shown)

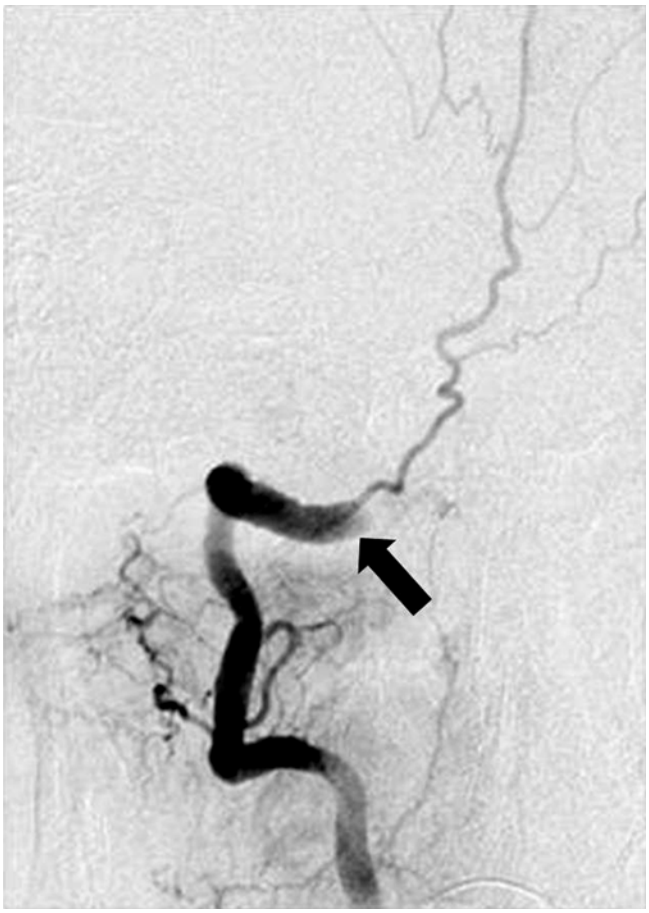

Fig. 11 Right VA DSA, AP view shows a blunt, rounded termination of flow with thinning of the contrast column (*arrow*), consistent with an acute occlusion. The posterior meningeal artery arises just proximal to the stump

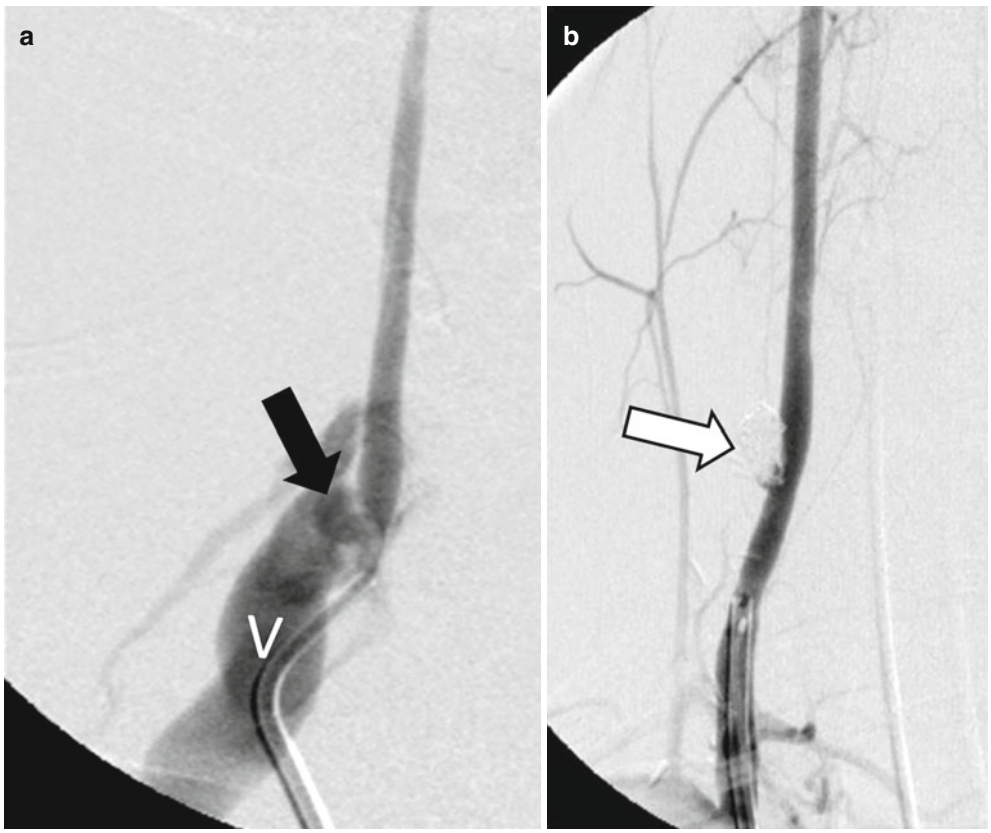

Fig. 12 Iatrogenic injury to the right vertebral artery during attempted central line placement resulted in a vertebral artery to vertebral venous fistula, seen on a right VA DSA (**a**, *arrow*). The fistulous connection is identified (*black arrow*), as well as rapid shunting into markedly enlarged vein (V). After closure of the fistula using platinum coils (**b**, *white arrow*), the vertebral artery fills normally, and the draining vein is no longer seen in the arterial phase. The white arrow on b is pointing to the coil ball, which is difficult to see because it is masked out

References

1. Berne JD, Reuland KS, Villarreal DH et al (2006) Sixteen slice multi-detector computed tomographic angiography improves the accuracy of screening for blunt cerebrovascular injury. J Trauma 60:1204–1209
2. Biffl WL, Moore EE, Offner PJ et al (2001) Blunt carotid and vertebral arterial injuries. World J Surg 25:1036–1043
3. Gdynia HJ, Kuhnlein P, Ludoph AC et al (2008) Connective tissue disorders in dissections of the carotid or vertebral arteries. J Clin Neurosci 15:489–494
4. Munera F, Danton G, Rivas LA et al (2009) Multidetector row computed tomography in the management of penetrating neck injuries. Semin Ultrasound CT MRI 30:195–204
5. Biffl WL, Ray CE, Moore EE, Franciose RJ et al (2002) Treatment-related outcomes from blunt cerebrovascular injuries: importance of routine follow-up arteriography. Ann Surg 235:699–706
6. Seth R, Obuchowski AM, Zoarski GH (2013) Endovascular repair of traumatic cervical internal carotid artery injuries: a safe and effective treatment option. AJNR Am J Neuroradiol 34(6):1219–1226
7. Anaya C, Munera F, Bloomer CW et al (2009) Screening multidetector computed tomographic angiography in the evaluation of blunt neck injuries: an evidence based approach. Semin Ultrasound CT MRI 30:205–214
8. Gardner DJ, Gosink BB, Kallman CE (1991) Internal carotid artery dissections: duplex ultrasound imaging. J Ultrasound Med 10:607–614
9. Montalvo BM, LeBlanq SD, Nunez DB Jr et al (1996) Color flow Doppler sonography in penetrating injuries of the neck. AJNR Am J Neuroradiol 17:943–951
10. Provenzale JM, Sarikaya B (2009) Comparison of test performance characteristics of MRI, MR angiography, and CT angiography in the diagnosis of carotid and vertebral artery dissection: a review of the medical literature. AJR Am J Roentgenol 193(4):1167–1174
11. Chokshi FH, Munera F, Rivas LA et al (2011) 64-MDCT angiography of blunt vascular injuries of the neck. AJR Am J Roentgenol 196(3):W309–W315