Clinical Presentation, Imaging, and Management of Complications due to Neurointerventional Procedures

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Abstract

Neurointervention is a rapidly evolving and complex field practiced by clinicians with backgrounds ranging from neurosurgery to radiology, neurology, cardiology, and vascular surgery. New devices, techniques, and clinical applications create exciting opportunities for impacting patient care, but also carry the potential for new iatrogenic injuries. Every step of every neurointerventional procedure carries risk, and a thorough appreciation of potential complications is fundamental to maximizing safety. This article presents the most frequent and dangerous iatrogenic injuries, their presentation, identification, prevention, and management.

Keywords

- interventional radiology
- neuroradiography
- cerebral angiography
- complications
- iatrogenic disorders

Objectives: Upon completion of this article, the reader will be able to discuss the clinical presentation, imaging, and management of complications arising from neurointerventional procedures.

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Adverse outcomes nearly always follow a cascade of minor deviations from standard practice, and seldom result from a single error.[1,2] In most cases a series of events must occur, in the correct order, at the correct time, for an iatrogenic injury to occur. Conversely, disasters may be avoided by identifying components of this cascade early on and initiating the appropriate response. Each neurointerventional procedure carries unique risks which, when anticipated, can frequently be avoided. The most frequent and dangerous of these risks are discussed below. The reader is encouraged to read pertinent sections of this article immediately prior to neurointerventional procedures.

Complications of Diagnostic Angiography

Cerebral Angiography

Diagnostic cerebral angiography remains the gold standard for imaging cerebral vasculature and is also the first step in performing neurointerventional procedures. The most common complications are thromboembolism and air embolism from catheters and wires resulting in cerebral ischemia. Disruption of atherosclerotic plaques and arterial dissection are additional potential causes of ischemic events. Ischemic stroke occurring due to cerebral angiography may take the form of a large vessel occlusion or a small, distal arterial branch occlusion. Recent large series have reported transient neurological complications ranging from 0 to 0.7% and permanent neurological complications in 0 to 0.5% of cerebral angiograms.[3–6] Quality improvement guidelines have recommended that transient neurologic deficits should occur in no more than 2.5% of patients and permanent neurologic deficits should occur in no more than 1% of patients.[7]

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Brain magnetic resonance imaging (MRI) done after cerebral angiography often demonstrates scattered focal regions of restricted diffusion, which are usually asymptomatic.[8] Symptomatic ischemic events occur with injury to eloquent cortex or critical deep structures, and may be observed on diffusion-weighted MRI imaging almost immediately following the event. Computed tomography (CT) angiography or repeat angiography may reveal disrupted atherosclerotic plaques or the classic "string sign" of an arterial dissection.[9] Effective management of ischemic stroke in this setting begins with prompt recognition of the event, either by detection of a neurological change in the patient or by identifying a freshly occluded artery on the angiogram. Intra-arterial thrombectomy or thrombolysis can then be undertaken. In situations in which a neurological deficit occurs in the postprocedure period after an angiogram, rapid imaging with CT or MRI may be helpful, followed by a return to the angiography suite for intra-arterial treatment if necessary.

Spinal Angiography

Diagnostic spinal angiography carries risks similar to cerebral angiography, such as spinal cord ischemic events from thromboembolism or air embolism resulting in myelopathy, as well as vessel dissection or disruption of atherosclerotic plaques. Transient myelopathy has been reported to occur from 0 to 2.2% of spinal angiography procedures; irreversible myelopathy is extremely rare.[10–12]

Nonneurologic Complications of Diagnostic Angiography

Contrast nephrotoxicity, arterial occlusion requiring surgical thrombectomy or thrombolysis, and development of arteriovenous fistula (AVF) or pseudoaneurysm occurs in ~0.2% diagnostic cerebral angiograms. Groin or retroperitoneal hematoma requiring either transfusion or surgical evacuation occurs in ~0.5% of procedures.[7]

Patients with connective tissue disorders such as Ehlers–Danlos are at heightened risk of complications such as vessel dissection and retroperitoneal hematoma with any neurointerventional procedure.[13,14]

Radiation exposure during neurointerventional procedures averages 1.67 mSv, with an estimated risk of death by radiation-induced cancer of 1 per 6,000 procedures.[15] Doses of 10 mSv or greater may occur in lengthy cases, with resultant hair loss.[16] Radiation exposure may be controlled by minimizing fluoroscopy time and the number of images acquired during angiograms, the use of dynamic acquisition, and virtual collimation.

Complications of Endovascular Treatment of Intracranial Aneurysms

Overall complication rates for coiling of intracranial aneurysms range from 8.4 to 18.9%.[17–20] Risk is increased in patients with subarachnoid hemorrhage, with the use of balloon- and stent-assisted coiling, treatment of very small and very large aneurysms,[21–25] and performance by inexperienced operators.[26]

Aneurysm or Vessel Perforation

The overall risk of vessel perforation is 4.1% during treatment of ruptured aneurysms and 0.5% with treatment of unruptured aneurysms.[27] Risk of perforation is higher during the treatment of aneurysms smaller than 4 mm.[28–30]

Aneurysm perforation during embolization may or may not be obvious on fluoroscopy or angiography. Other clues to the occurrence of a perforation are a sudden elevation in intracranial pressure or blood pressure, bradycardia, or a prolonged sinus pause. In awake patients, a sudden headache or a neurological change may signal a perforation. Whenever a perforation is identified or suspected, the perforating wire, catheter, or coil should be left in position and a prompt guide catheter angiogram should be done. Active extravasation of contrast may not be observed if the perforation is occluded by the microcatheter or microwire. A CT scan performed post-perforation will appear to show more subarachnoid blood than is actually present due to the presence of contrast material in the subarachnoid space (►**Figs. 1** and **2**).

Following vessel perforation, heparin anticoagulation should be reversed with protamine (dose: 1 mg of protamine for every 100 units of heparin given). When possible, coils may be deployed to treat the perforation. Alternatively, a second microcatheter can be used to continue coiling the aneurysm while the first microcatheter is left in place.[31] Another strategy is to use a balloon catheter to occlude the parent vessel to reduce the bleeding. In some cases, parent vessel sacrifice by embolization may be necessary to stop the hemorrhage.

Thromboembolism

Thrombus formation can result from manipulation of catheters, wires, coils, or balloons.[32] Symptomatic thromboembolism occurs in 2 to 8% of patients during aneurysm coiling, but

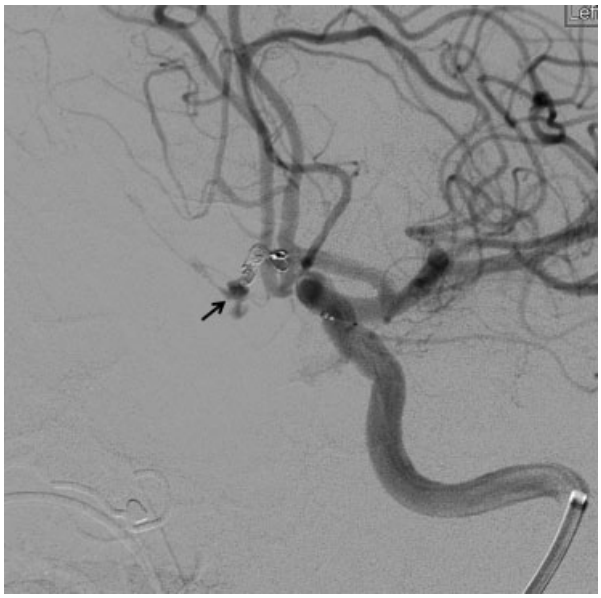

Fig. 1 Digital subtraction angiogram during coiling of a ruptured anterior communicating artery aneurysm. Contrast extravasation into the subarachnoid space is noted from the aneurysm dome due to perforation by a coil (*arrow*).

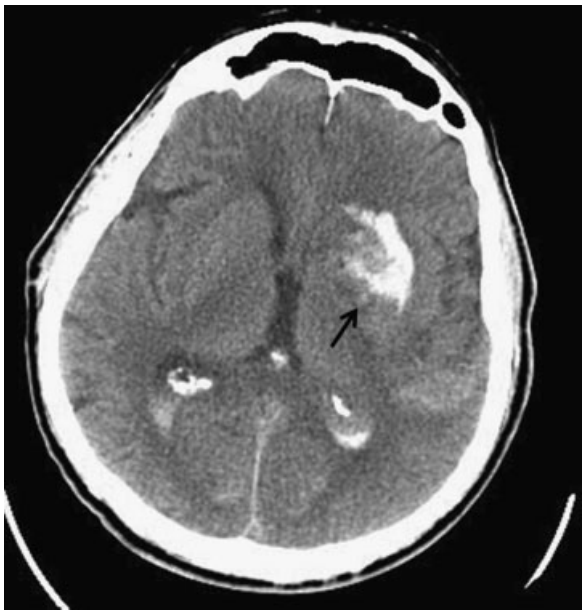

Fig. 2 Axial noncontrast CT scan following thrombectomy for internal carotid artery occlusion and ischemic stroke. A "contrastoma" has appeared in the left thalamus due to extravasation of contrast material secondary to blood–brain barrier breakdown (*arrow*).

the majority of these are transient.[32–38] Thrombi may be directly visualized with guide catheter angiogram as a filling defect within the parent vessel, or as a distal vessel occlusion. Adjustment of the angiogram image during the capillary phase by increasing the contrast and reducing the brightness at the monitor (a so-called stroke-o-gram) will make the ischemic defect more apparent and help identify the territory of affected arteries.

Thrombus formation during coiling is primarily mediated by platelet aggregation. Abciximab and other antiplatelet agents are the first step in management. Partial dosing of abciximab can have a paradoxical platelet activation effect, and therefore a full loading dose followed by intravenous (IV) infusion for 12 hours is recommended.[39,40] Additionally, mechanical thrombectomy can be attempted using a 2- or 4-mm snare.[41] Due to high rates of hemorrhagic complications, thrombolytic agents should be avoided, particularly in cases of ruptured aneurysms.[42]

Coil Dislodgement and Embolization

An appropriately sized initial framing coil is essential for successful embolization. An unstable or malpositioned coil can herniate into the parent vessel or embolize distally. Overpacking of an aneurysm can also dislodge previously released coils. To reduce this risk, shorter and softer coils are recommended in the final stages of coiling procedures.

Coil herniation places the patient at increased risk of flow compromise or thrombus formation in the parent vessel, while coil migration can result in distal ischemia. Wide-necked aneurysms are at greatest risk of coil dislodgement, with an overall incidence of 0.5%.[20] A variety of devices are available for retrieving detached coils. Stent or balloon assistance may also be used to reinsert herniated coils into the aneurysm[43,44]; if the coil cannot be safely retrieved or repacked into the aneurysm, long-term antiplatelet therapy is recommended.

Coil Stretching and Unraveling

When the distal portion of a coil becomes trapped inside an aneurysm, attempted withdrawal of the coil can lead to stretching or unraveling. A slightly stretched coil, one that is only slightly lengthened, may still be deployed using stent or balloon assistance.[44,45] A microsnare can also be used to withdraw a stretched coil.[46] By contrast, a completely unraveled coil may elongate to over 1 m in length and can be difficult to control. A lengthy elongated coil may be withdrawn to the level of the femoral artery and secured to the vessel wall.[47] Alternatively, the coil can be partially withdrawn, redirected, and released into an extracranial artery, such as a branch of the external carotid artery.

Vessel Injury

Arterial injury due to wire or guide catheter–induced intimal damage occurs in 0.6 to 3.6% of coiling cases.[19,20,48] Intimal injury is likely underreported, as many operators do not routinely perform a surveillance angiogram of the access vessel after coiling. The vertebral artery is thought to be at greater risk of injury than the internal carotid.[4,49,50] Vessel dissection is generally asymptomatic, but places the patient at increased risk of occlusive or thromboembolic complications.[51] Imaging may directly reveal an injury in the form of a double lumen or intimal flap in arterial dissection, or indirectly in the form of arterial occlusion or stenosis, string sign, aneurysm, or pseudoaneurysm. Initial management of arterial dissection consists of dual antiplatelet therapy with aspirin 325 mg and clopidogrel 75 mg daily.[52] For flow-limiting injury, anticoagulation with IV heparin or stenting may be necessary.[53] Follow-up imaging should be done at 3 to 6 months, as 90% of dissections causing stenosis will be found to have interval resolution, and up to 50% of dissections causing occlusion will be found to have interval recanalization.[54] Dual antiplatelet therapy may be discontinued at that time.

Rehemorrhage

Rehemorrhage after coiling of ruptured intracranial aneurysms occurs in some 0.9% of cases within 30 days after the procedure.[55] Early rehemorrhage usually occurs in aneurysms that were incompletely occluded after initial treatment. Late rehemorrhage typically occurs in aneurysms with recanalization,[56,57] underscoring the need for routine surveillance of coiled aneurysms.

Flow Diverters and Stent-Assisted Coiling

Flow diverters are wire mesh stents used to disrupt flow into an aneurysm, leading to thrombosis and endothelialization across the aneurysm neck. These devices are used for the management of aneurysms that are not amenable to traditional endosaccular approaches. The use of Pipeline flow diversion (ev3; Plymouth, MN) carried a 5.6% risk of major ipsilateral stroke or neurologic death.[58] Risk of parent vessel

or branch occlusion, as well as delayed aneurysmal hemorrhage, is increased in the setting of inadequate antiplatelet therapy.[59,60] Ipsilateral intraparenchymal hemorrhage has been reported in 3% of cases,[61] and delayed rupture of the treated aneurysm has been observed in 2.1% of cases in which the aneurysm is >10 mm in size.[62,63] The occurrence of delayed hemorrhage in larger aneurysms treated with flow diversion has led to the hypothesis that an inflammatory response develops after acute thrombosis of the aneurysm, leading to erosion and rupture of the aneurysm in some cases. Because of this, coil embolization in addition to flow diversion has been recommended for larger aneurysms.[60–66]

Complications of Endovascular Treatment of Acute Ischemic Stroke

IV infusion of alteplase is considered to be first-line treatment for most patients with acute ischemic stroke presenting within 3 to 4.5 hours of onset of symptoms. Patients with large vessel occlusions and patients who are not candidates for IV alteplase are candidates for intra-arterial thrombectomy.

Mechanical Thrombectomy for Acute Ischemic Stroke

Intra-arterial treatment can be an effective alternative for patients who cannot be treated with IV alteplase, or do not respond to treatment with IV alteplase. The most commonly used intra-arterial techniques are suction thrombectomy and stent-retriever (*stentriever*) thrombectomy. Contemporary overall complication rates with thrombectomy for ischemic stroke include procedure-related mortality ranging from 0 to 2% and permanent neurological injury of 3 to 6.5%.[67–69] The main threat after mechanical thrombectomy is intracerebral hemorrhage, which occurs in 4.9 to 10% of cases.[67–69] It is common to see small regions of contrast extravasation in the affected territory of the brain after mechanical thrombectomy; this should not be confused with intracerebral hemorrhage. Tips for avoiding complications with intra-arterial treatment of ischemic stroke include meticulous attention to the patient's neurological status during and after the procedure, reversal of heparin for bleeding complications, and adequate control of blood pressure. Although specific guidelines for the management of blood pressure in this setting have not yet been published, adherence to the American Heart Association Guidelines recommendation to maintain blood pressure < 180/105 mm Hg (for patients treated with IV alteplase) seems reasonable.[33]

Because patients undergoing intra-arterial treatment of ischemic stroke have also often received IV alteplase, an uncommon but significant complication of alteplase merits consideration. Anaphylactoid reactions and angioedema are well-recognized reactions to alteplase and other thrombolytic agents, occurring in up to 2% of patients.[70] Angioedema presents with localized swelling of the tongue, lips, or oropharynx, and typically occurs within 6 hours of exposure. Patients on angiotensin-converting-enzyme inhibitors appear to be at increased risk.[71] While symptoms are typically mild, life-threatening airway obstruction may occur; elective oropharyngeal intubation for airway control may be preferred. CT scan of the face is also recommended to rule out tongue or oropharyngeal hemorrhage. High-dose dexamethasone (10 mg IV bolus, then 6 mg every 6 hours) and antihistamines may speed up resolution of the angioedema, while aerosolized epinephrine can reduce laryngeal edema.[72]

Complications of Extracranial Carotid Angioplasty and Stenting

While carotid endarterectomy (CEA) remains the gold standard for treatment of uncomplicated carotid stenosis, carotid angioplasty and stenting (CAS) is a viable option for select patients. The CREST trial found that both treatment options showed durable protection against ipsilateral stroke, with a higher rate of periprocedural stroke during CAS and a higher rate of myocardial infarction following CEA.[73] Thromboembolism, dissection, intracranial hemorrhage (ICH), hyperperfusion syndrome, bradycardia, and hypotension are all potential complications of CAS.

The occurrence of an acute neurological change during the procedure suggests thromboembolism, and should be followed by an intracranial angiogram. The angiogram may demonstrate intraluminal thrombus or delayed contrast passage through distal intracranial vessels, indicating a shower of emboli. When thromboemboli are identified, options include mechanical thrombectomy or infusion of an IV glycoprotein IIb/IIIa inhibitors such as abciximab. Abciximab has an advantage over other glycoprotein IIb/IIIa inhibitors in that it can be reversed with platelet transfusion.[74] Intra-arterial thrombolytics can also be used, but may be less effective against the platelet-rich thrombi, and also carry a risk of ICH.

Angioplasty is the maneuver during CAS procedures most associated with thromboembolic complications[75]; therefore, minimal angioplasty is often prudent, and excessive dilation should be avoided as it also places the patient at risk of bradycardia and hypotension.[76] The operator should be prepared for the possibility of bradycardia and hypotension by having IV atropine and dopamine prepared and ready to inject immediately after angioplasty, particularly when dilating at the level of the carotid sinus.

ICH can also complicate CAS. A new neurologic deficit, headache, and a Cushing response (hypertension and bradycardia) should raise suspicion for ICH. Immediate head CT should be performed, and the sheath should be left in place. If ICH is identified, heparin anticoagulation is reversed with protamine, and strict blood pressure control is maintained.

Hyperperfusion syndrome can occur in up to 5% of CAS cases,[77] and is associated with ICH in 0.7%.[78] Patients present with ipsilateral headache, nausea, seizures, or focal neurologic deficit without radiographic evidence of ischemia from 6 hours to 4 days following CAS. The condition is theorized to result from an abrupt increase in cerebral blood flow following CAS in the setting of previous chronic hypoperfusion and impaired autoregulation. Treatment consists of strict blood pressure control, and typically resolves without permanent deficit in the absence of associated ICH. Hyperperfusion syndrome must be distinguished from transient contrast

encephalopathy, another complication rarely seen after CAS.[79] In transient contrast encephalopathy, CT will show cortical enhancement and cerebral edema. The syndrome is typically self-limited and most patients have no long-term deficits.

Approximately 30% of cerebral ischemic events occur in a delayed fashion, 2 to 14 days following the procedure.[80,81] For these patients, initial evaluation begins with noncontrast head CT and carotid duplex exam. When neurologic deficits suggest a large-vessel occlusion, emergent return to the angiography suite for thrombectomy or intra-arterial thrombolysis should be considered.[81] Recurrent focal neurologic deficit months or years after CAS suggests in-stent restenosis, which may be treated using a cutting balloon with distal embolic protection.[82,83]

Complications of Intracranial Angioplasty and Stenting

This section concerns procedures for the treatment of intracranial atherosclerotic stenosis and vasospasm. Stent-assisted treatment of intracranial aneurysms is also discussed in the section Complications of endovascular treatment of intracranial aneurysms.

Patients with symptomatic intracranial stenosis greater than 70% who have failed medical therapy may be candidates for balloon angioplasty with or without stenting. No Class I evidence yet exists that shows a benefit of intracranial angioplasty and stenting over medical management, and as such, patient selection is paramount. The overall perioperative stroke rate has been estimated at 7.9%, with a perioperative death rate of 3.4%.[84] Vessel perforation, intraluminal thrombus, shower emboli, and dissection are also potential complications, and angioplasty without stenting carries a 30% rate of restenosis at 3 and 12 months.[85] Tips for the avoidance of complications during intracranial stenosis include minimalistic angioplasty to avoid artery rupture and dissection, and using angioplasty alone without placement of a stent if the postangioplasty angiogram shows significant improvement in the stenosis.

Treatment of Vasospasm

Cerebral vasospasm presents most commonly following aneurysmal subarachnoid hemorrhage, but may also complicate neurointerventional procedures. Hyperdynamic therapy, intra-arterial injection of pharmacologic agents, and balloon angioplasty are all potential therapeutic interventions. Some investigators recommend angioplasty as first-line treatment for vasospasm on an emergent basis,[86] while others advocate a trial of hyperdynamic therapy prior to proceeding with angioplasty.[87,88] Balloon angioplasty is used for symptomatic vasospasm affecting intracranial arteries >1.5 mm in diameter,[89] which includes the intracranial internal carotid artery (ICA), vertebral and basilar arteries, and the M1, A1, and P1 segments. For vessels that are not easily accessible or safely treated with a balloon, intra-arterial injection of nicardipine or verapamil can be used.[90]

In subarachnoid hemorrhage patients who have undergone craniotomy, procedural systemic heparinization poses a 1.8% risk of major hemorrhage.[91] By contrast, use of systemic heparinization is thought generally safe in subarachnoid hemorrhage patients with a ventriculostomy.[92,93] The major complication rate with endovascular treatment for vasospasm was recently reported at 5%, with a 1.1% incidence of vessel rupture.[94] Other complications are discussed in greater detail above, and include thromboembolism, arterial dissection, reperfusion hemorrhage, branch occlusion, bleeding from untreated aneurysms, retroperitoneal hemorrhage, and groin hematoma.[95] Tips for the avoidance of complications with intracranial angioplasty for vasospasm include limiting treatment to only arteries that are relatively easy to access with a balloon catheter and undersizing the balloon diameter to minimize risk of rupture.

Complications of Embolization Procedures

Endovascular embolization is a versatile technique used in the management of intracranial and spinal tumors, arteriovenous malformations (AVMs), and AVFs. A wide range of embolic materials and techniques are used, each of which carries its own set of potential complications.

Retained Microcatheter

Microcatheters may become trapped by both adhesive and nonadhesive liquid embolic material. A retained microcatheter may be retrieved by administering continuous, gentle traction on the device for 5 to 10 minutes or more. Additional techniques for the retrieval of a trapped microcatheter include using a monorail snare technique to grasp the trapped catheter[96] or advancing an intermediate catheter over the trapped microcatheter to apply countertraction.[97] Firmly glued-in catheters may need to be permanently implanted by cutting the catheter at the level of the femoral artery.[98] Blood flow down the aorta will press the microcatheter against the arterial wall, allowing it to become endothelialized over time.[99] Patients should be kept on aspirin indefinitely if a retained microcatheter is left in place. Both femoral artery pseudoaneurysm and popliteal artery occlusion have been reported after cutting and leaving in place a glued-in catheter.[100,101] If the catheter impairs cerebral blood flow, surgical extraction may be required.

Microcatheter fractures above the level of the aorta may result in distal embolization and infarction.[102] In these cases, a microsnare can be used to grasp the fragment, or a self-expanding stent can be used to trap the microcatheter against the parent artery wall. If a stent is used, dual antiplatelet therapy is required.

Hemorrhage

Hemorrhage of tumors can occur during or after embolization, and is usually due to infarction of the tumor with hemorrhagic transformation. Risk of tumor hemorrhage can be minimized by avoiding overly ambitious embolization of large tumors. Operators should be on the alert for the possibility of hemorrhage, particularly after the procedure, and prepare for the possibility of urgent craniotomy and resection of the tumor if necessary. AVM rupture can occur

during or after embolization due to occlusion of draining veins without adequate disruption of feeding arterial flow,[103] with reported rates varying from 1.6 to 10.6%.[104–106] Importantly, medium and large brain AVMs should not be completely embolized in a single session, as complete occlusion of these lesions carries a higher risk of hemorrhage.[105]

Postembolization Edema or Hemorrhage

Significant cerebral edema may occur following occlusion of high-flow fistulas, AVMs, pial AVFs, or tumors; this is the so-called normal perfusion breakthrough syndrome.[107] Abrupt occlusion of a longstanding arteriovenous shunt exposes vessels with disturbed autoregulation to increased flow, resulting in hyperemic cerebral edema and the potential for hemorrhage. Patients who are thought to be at risk for postprocedural cerebral edema may be treated with dexamethasone before and after the procedure; in addition, indomethacin may be used to induce cerebral vasoconstriction.[108] Strict control of blood pressure is essential, as is aggressive treatment of intracranial hypertension when a ventriculostomy is in place.

Thromboembolism

Rates of thromboembolic complications vary with the embolic agent used. A recent study demonstrated a 3.8% risk of stroke in AVM embolization using *n*-BCA, versus a 5.8% risk of stroke following PVA embolization.[109] Identification and management of thromboembolism with endovascular procedures was discussed above in the Complications of endovascular treatment of intracranial aneurysms section.

Embolization of Unintended Targets

Inadvertent embolization of the brain, retina, or cranial nerves can occur through several different mechanisms. First, meticulous effort must be made to identify potential dangerous collaterals prior to embolization; a superselective, high-magnification angiogram to identify collaterals to the brain or eye is essential, and provocative testing should be done whenever possible. Reflux of embolic material in a retrograde direction may occur during embolization, causing the embolic material to spill into collateral vessels. The risk of reflux can be minimized by mixing the embolic material with iodinated contrast material and doing the injection under fluoroscopic guidance; the injection should be stopped and aspiration through the microcatheter should be performed whenever reflux of contrast material is observed. Inadvertent embolization may also occur during treatment of high-flow lesions, due to the embolic agent traveling more distally than intended. Smaller particles may penetrate more deeply into tumors and cause greater devascularization, but are also associated with higher hemorrhage rates.[110,111] Intratumoral shunting also increases the risk of unintended distal embolization. For extracranial embolizations, close attention must be paid to known extracranial–intracranial anastomoses to prevent embolic stroke. Cranial nerve palsies may occur via inadvertent embolization of the vasa nervorum, and are a risk with both intracranial and extracranial embolization procedures.[112] Embolic agents may travel to the pulmonary vasculature,[113–115] and embolization of superficial vessels in the head and neck can result in ischemic necrosis of skin and mucosa. Retinal embolization has also been reported after embolization of meningiomas.[116] In general, when using particle embolic material, the use of larger particles (>300-μm diameter) may help minimize risk of inadvertent embolization because the particles are too large to pass via collaterals into many vital end-organ arteries.

Complications of Testing Procedures

Balloon Test Occlusion

Balloon test occlusion is a commonly used technique to assess the risk of cerebral ischemia prior to arterial sacrifice. When performed without prior test occlusion, sacrifice of the ICA carries a 26% stroke rate and a mortality rate of 12%[117]; by contrast, internal carotid occlusion after test occlusion carried a stroke rate of 16.2% in a recent series.[118] The high stroke rate following a negative balloon occlusion test indicates the need for adjunctive testing prior to vessel sacrifice. Neurologic complications following balloon test occlusion include thromboembolic stroke in 0.4% of cases, arterial dissection in 1.2%, and aneurysm dissection in 0.2%.[119] Very rarely, overinflation of the balloon can rupture intracranial vessels or result in development of a carotid-cavernous fistula. Balloon inflation in the carotid bulb or basilar artery may also cause a vasovagal response with bradycardia and hypotension, or rarely, cardiac arrest. The vast majority of vasovagal responses respond quickly to balloon deflation, and do not require IV fluid bolus or vasopressor administration.

WADA Testing

Intra-arterial injection of amobarbital or methohexital is a standard component of the preoperative work-up for epilepsy in an attempt to lateralize speech and memory function. Thromboembolism and arterial dissection with subsequent occlusion or pseudoaneurysm occur with similar frequencies as diagnostic cerebral angiograms. Inadvertent Amytal injection into the basilar artery can occur in patients with carotidobasilar anastomoses such as persistent trigeminal artery; posterior circulation injection of Amytal typically results in apnea and unconsciousness. Seizures and cerebral edema are rare complications of WADA testing.[120]

Complications of Venous Procedures

Venous Sampling

The most common indication for intracranial venous sampling is in the evaluation of Cushing disease. Inferior petrosal sinus sampling (IPSS) may provide confirmation and lateralization of an adrenocorticotropic hormone–secreting pituitary adenoma when biochemical testing and MRI do not clearly identify the source. Permanent neurologic complications at experienced centers are very rare, occurring in fewer than 0.1% of cases.[121] Reported complications include brainstem ischemia or hemorrhage,[122] subarachnoid hemorrhage,[123] and transient sixth cranial nerve palsy.[124] In one series, 2 out of 34 (5.9%) patients undergoing IPSS had deep

venous thrombosis, and 1 patient died from resultant pulmonary embolism.[125]

Transvenous Embolization

Transvenous embolization is a useful technique for selected carotid-cavernous fistulas, dural AVFs, vein of Galen malformations, and vertebral-venous fistulas. Risks include perforation or rupture of intracranial vessels, venous infarction, and worsening of venous hypertension and are discussed above. Additionally, transvenous liquid embolics can reflux into arterial feeders and further reflux into normal arterial territories, causing ischemia of normal tissue. Intracranial abscess has also been reported after transvenous embolization.[126] Transient neurologic complications may be noted in up to 10% of patients, although permanent deficits are rare.[127] Even after successful transvenous embolization of a dural fistula, delayed development of a second fistula at a different site can still occur.[128,129]

Venous Thrombolysis and Thrombectomy

Cerebral venous sinus thrombosis has a high associated mortality rate. Patients with progressive neurologic deterioration, despite systemic anticoagulation, may benefit from endovascular therapy.[130,131] Mechanical thrombectomy and infusion of thrombolytics are standard techniques. Accurate complication rates surrounding venous thrombolysis and thrombectomy are lacking due to the small sizes of reported series. Worsening of cerebral edema or hemorrhage, venous perforation and ICH, and rethrombosis of revascularized venous structures have all been reported. Additionally, mechanical clot disruption in the larger dural venous sinuses and jugular veins may cause symptomatic pulmonary emboli.

Conclusion

New and rapidly changing tools and techniques allow for neurointerventional management of a wide range of neurologic conditions. Embedded within this impressive flexibility are new risks which, in many cases, are still poorly characterized. Full appreciation for potential iatrogenic injury is essential in identifying and modifying dangerous situations and maximizing patient safety.

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