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Preoperative Embolization of Central Nervous System Tumors

Vivek R. Deshmukh, MD, David J. Fiorella, MD, Cameron G. McDougall, MD, Robert F. Spetzler, MD, Felipe C. Albuquerque, MD*

Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph's Hospital and Medical Center, 350 West Thomas Road, Phoenix, AZ 85013, USA

Surgical excision of hypervascular central nervous system (CNS) tumors can be daunting and is associated with the potential for staggering blood loss. The need to mitigate intraoperative tumoral hemorrhage has fostered the refinement of neuroendovascular techniques for preoperative embolization of tumors. Since these techniques were first described in the early 1970s, the field has matured rapidly, with advances in microcatheter technology and ongoing improvements in the design of embolisates. Nonetheless, the tenets fundamental to embolization of tumors are not novel. They include an understanding of neurovascular anatomy and of the natural history and behavior of tumors.

Principles of embolization

Tumor embolization is defined as the interruption of blood supply to a tumor. The goal of preoperative embolization is to decrease intraoperative blood loss. Embolization-induced tumor ischemia often softens a tumor, thereby facilitating its resection and reducing the compression exerted on nearby neural structures. Theoretically, tumor resection then becomes safer and more complete. Other purported goals of embolization are to reduce operating time, to relieve intractable pain, and to decrease the likelihood of a recurrence.

Pre-embolization procedure

If a patient is identified as having a hypervascular tumor on preoperative MRI, the surgical team should discuss the utility of preoperative embolization. In many cases, surgical resection may not be undertaken safely without embolization and vice versa. The tumor surgeon and the neuroendovascular surgeon must agree on the goals of embolization and on the degree of aggressiveness required. These goals are tailored to each patient.

Embolization procedure

Embolization may be transarterial via a transfemoral route or via direct puncture of the tumor. Embolization may be performed in a single session, or it may be staged. Typically, patients undergo angiography and particulate embolization under conscious sedation. Conscious sedation permits provocative testing of the cranial nerves and balloon test occlusion of the carotid artery as needed. The location of the tumor dictates the vessels to be examined. A thorough examination of the external carotid artery (ECA) is mandatory. If N-butyl cyanoacrylate (NBCA) glue is used, the patient may be placed under general anesthesia or conscious sedation. We prefer general anesthesia, because the hazards associated with patient movement are magnified during glue embolization. Somatosensory evoked potentials and

* Corresponding author. c/o Neuroscience Publications, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013, USA.
E-mail address: neuropub@chw.edu (F.C. Albuquerque).

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electroencephalography are monitored routinely by our group.

The size of the femoral sheath should be tailored to the surgical goals, and attention should be given to the necessity of a balloon delivery system. A coaxial Envoy (Cordis, Miami, Florida) guiding catheter and a UCSF-3 (Cordis) catheter are advanced to the ECA or internal carotid artery (ICA). The UCSF-3 catheter is removed or steered selectively into an ECA branch to provide further support of the microcatheter system.

Once an embolizable arterial supply is identified, a microcatheter is advanced coaxially through the guiding catheter to the feeding artery with the use of fluoroscopic roadmap guidance. The microcatheter is navigated to the most distal point. We prefer using the new-generation hydrophilic microcatheters and microwires (wire diameter is typically 0.014 or 0.010 mm). Embolization is performed with polyvinyl alcohol (PVA) particles or NBCA glue embolisate.

Before embolization begins, several anatomic factors should be assessed: the presence of extracranial-intracranial (EC-IC) anastomoses, shared arterial supply between tumor and normal structures, the caliber of tumor vessels, and the potential for embolisate to reflux into the parent vessel. These factors are best evaluated by careful superselective microcatheter angiography. Embolization is most beneficial for tumors primarily supplied by the ECA [1,2]. For particulates, the number of emboli that reach a vascular tumor depends on the blood flow to the lesion relative to the rest of the brain. Therefore, selective catheterization allows a maximal amount of embolisate to be delivered to the lesion.

Occlusion of the proximal feeding arteries only reduces blood flow to the tumor temporarily. Collateral supply quickly develops, and the beneficial effect of the embolization is short-lived. Occlusion of proximal arteries may, however, be considered if access to distal supply is poor. The logical end point for embolization is an angiographically demonstrated decrease in the vascularity of the tumor with preservation of normal arterial territories. The ideal barometers for efficacy of embolization include MRI-verified tumor infarction, histologic evidence of tumor necrosis, ease of surgical resection, and mitigation of intraoperative blood loss.

The introduction of variable stiffness and hydrophilic microcatheters has permitted microvascular selectivity. For tumors with significant blood supply from the cavernous ICA, embolization can be performed via direct microcatheterization of these small branches. If technically infeasible, another option [3,4] (used by C.G.M) is occlusion of the distal ICA with a nondetachable balloon and subsequent infusion of embolic material (typically PVA or ethanol) into the segment of the ICA supplying the tumor. Before the balloon is deflated, this region of the ICA must be flushed with heparinized saline (at least 20 mL). A similar volume is withdrawn from the guiding catheter to irrigate the ICA. Theron et al [4] believe that this balloon-assisted technique may be used for hypervascular lesions supplied by branches of the vertebral artery (ie, posterior inferior cerebellar artery [PICA]). Garcia-Cervinon et al [5], however, described four patients who developed balloon-related complications (balloon displacement/migration and failed balloon deflation). None of these patients suffered a permanent neurologic complication. Balloon-assisted embolization must be used judiciously because of the distal migration of embolic material on balloon deflation and because of potential vessel endothelial injury from ethanol.

Pial vessels are fragile and are thus associated with a high risk of arterial perforation and intracranial hemorrhage. The potential for stroke from thromboembolism within pial vessels is also higher than the risk of thromboembolic complications within the circulation of the ECA.

When a hypervascular tumor is supplied by the ophthalmic artery, embolization may be performed safely if a few principles are followed. First, embolization must be performed only if it is essential and only if the embolized vessel cannot readily be accessed surgically. The central retinal artery and choroidal blush must be visualized. Embolisates must not be allowed to enter the central retinal artery. A lidocaine or Amytal (sodium amobarbital) test may be performed to verify that occlusion of ophthalmic artery branches will not cause blindness. If the central retinal artery is thought to be at high risk, coils or larger diameter embolisates (>400 μm) may be used. Their use does not guarantee the integrity of the central retinal artery, however. Lefkowitz et al [6] embolized three anterior skull base meningiomas and one nasal angiofibroma via the ophthalmic artery. One patient experienced transient visual deterioration after embolization with PVA (700–1000-μm particles).

Intraoperative percutaneous tumor puncture has been advocated for tumors predominantly

supplied by the ICA or pial vessels. Patients whose ECA has already been sacrificed are perhaps better served with direct tumor puncture. Casasco et al [7] recommended the use of intraoperative fluoroscopy to assist with the embolization. If reflux into the feeding arteries is visualized, embolization should cease. They also advocated using this technique for palliation in patients with inoperable tumors or for the treatment of elderly or debilitated patients. For lesions not readily amenable to endovascular therapy, George et al [8] delivered NBCA embolisate before or during surgery directly into superficial tumors with a standard 18- or 19-gauge needle and directly into deeply seated tumors with a 16-gauge Teflon sheath needle. Of 21 patients with tumors, such as juvenile nasopharyngeal angiofibromas, hemangiopericytomas, or metastases, who were treated with this technique, no patient suffered a permanent neurologic complication.

Postembolization procedure

After embolization, patients should be monitored in an intensive care unit for 24 hours. Particular attention must be paid to periprocedural stroke, cranial nerve deficits, or intracranial hemorrhage. Corticosteroids are recommended for large tumors associated with significant surrounding edema. Postembolization MRI is often obtained for the purpose of frameless stereotaxy. These images should be reviewed for intracranial hemorrhage or exacerbation of peritumoral edema. Such findings may warrant more urgent surgical excision.

Radiographic effects of embolization

Radiographic demonstration of tumor necrosis after particulate embolization seems to be indiscriminate. Terada et al [9] noted disappearance of tumor flow voids after embolization with Gelfoam or PVA. Most tumors showed a decrease in the level of contrast enhancement. Bendszus et al [10] found that more completely embolized tumors showed less contrast enhancement on postembolization MRI. Wakhloo et al [11], however, reported diminished enhancement on contrast MRI in only 2 of 14 patients after embolization with 150- to 300-μm particulates. They theorized that this disappointing radiographic result reflected proximal occlusion of the feeding arteries by larger particulates. Grand et al [12] described only a minimal decrease in tumor enhancement after embolization and attributed this discrepancy to vasospasm of the embolized vessels. Therefore, smaller particulates may allow embolization of more distal capillaries, increasing the likelihood of tumor necrosis after embolization.

Timing of embolization

The optimal timing for preoperative embolization is controversial [13–15]. Some authors recommend surgical resection 1 to 5 days after embolization [15–17], whereas others advocate waiting 1 to 2 weeks [13]. With time, embolization-induced necrosis shrinks and softens the tumor, thereby facilitating surgical resection. Compared with more proximal occlusion, delivering microemboli directly into the tumor maximizes this necrotic effect. The potential for recanalization of embolized vessels and collateral development increases if surgical resection is significantly delayed (>1 week). In contrast, Kai et al [18] retrospectively reviewed 45 patients with meningiomas embolized with cellulose porous beads. Resectability was greatest in tumors excised 7 to 9 days after embolization. The consensus remains, however, that the greatest benefit is derived from embolization if surgical resection is performed within a few days of embolization [2,17,19].

Embolisates

The ideal embolic agent should be permanent, easily deployed, and should not encumber tumor resection. Historically, various embolic agents have been used for the preoperative embolization of tumors. These permanent or temporary embolic agents include liquid tissue adhesives, such as NBCA and silicone rubber, and particulates, such as silastic beads, lyophilized dura mater, phenytoin, microspheres [20], microfibrillar collagen [21], oxidized cellulose, gelatin sponge, PVA [22], and fibrin glue [14]. Ethanol and detachable coils should also be included in this armamentarium.

Polyvinyl alcohol

PVA is the most common particulate agent used. It can be used for all tumors, including those with associated with high blood flow and arteriovenous shunting. PVA is shaved into precisely sized particles from an original block form [23–25]. The size of particles can range from 50 to 150 μm to 500 to 1200 μm.

Large embolisates do not penetrate deeply into tumor tissue, but they pose less risk of embolizing adjacent normal tissue. Smaller particulates and liquid agents penetrate deeply into the tumor but also have the potential to migrate into normal territory. PVA is delivered until stasis of contrast is noted in the embolized vessel. Should any reflux be visible, embolization should be discontinued.

PVA has several advantages. The particles are inert and water insoluble. They are absorbed slowly and are known to expand to occlude arteries with a diameter larger than the inner diameter of the microcatheter. They produce a vigorous inflammatory reaction. Polymorphonuclear proteins predominate at 2 weeks; a giant cell reaction occurs at 3 months; and an adherent, organized, partially calcified thrombus appears at 9 months [24,26]. PVA embolisate is long lasting but not permanent. Degradation occurs within several weeks to months. Technically, PVA is easy to deliver. The risk of stroke or cranial nerve palsy with the use of PVA particles diminishes as larger particles are used. PVA, however, has limitations. Early recanalization can occur when thrombus between the particles is dissolved by endogenous lytics. Furthermore, the high friction coefficient of PVA can lead to frequent catheter obstruction.

Tissue adhesive embolisate

NBCA glue embolisate is a stable, nonabsorbable, liquid polymerizing agent that is commonly used to treat arteriovenous malformations (AVMs) or fistulas and, less frequently, tumors. NBCA is mixed with an oil-based contrast agent (Ethiodol [ethiodized oil]), usually at a ratio of 1 mL to 2.5 mL. This formulation polymerizes immediately on contact with ionic solution or blood and occludes distal and proximal feeders almost simultaneously.

NBCA is a more permanent agent than PVA, and it immediately obliterates the feeding branch. NBCA is used with tumors less often than PVA because of its tendency to occlude distal and proximal vessels, which may be associated with a greater likelihood of cranial nerve injury or stroke. The delivery of NBCA has technical constraints that require experience to use it successfully. When used for ophthalmic lesions, NBCA can cause ocular myositis, resulting in ocular pain [6]. If acrylic glue is used for embolization, microcatheters can get caught or break, particularly if the microcatheter is bathed in the glue for an inordinate length of time [27].

Cellulose beads and microspheres

Cellulose porous beads also have been used for the preoperative embolization of tumors. Their favorable properties include a uniform size, a specific gravity similar to that of blood, and a positive charge that prevents clumping. Hamada et al [28] performed a prospective clinical trial in which 16 patients (13 with tumors) underwent embolization with cellulose porous beads measuring 150 or 200 μm. None of the patients suffered complications. Postembolization angiography showed satisfactory stasis in all cases. Histologic analysis of embolized vessels demonstrated no stretching of the embolized vessel and only mild inflammatory reactions. Trisacryl gelatin microspheres are a hydrophilic, nonabsorbable, collagen-coated agent, which is calibrated, deformable, and tends not to aggregate. Progressively larger embospheres may be used in the course of embolization. Bendszus et al [29] compared trisacryl gelatin microspheres with PVA particles in achieving distal microembolization and in mitigating intraoperative blood loss. Their most important finding was that the microspheres penetrated more distally than PVA particles. Further investigations are needed to establish the role of cellulose beads and microspheres in this arena.

Alcohol

Alcohol is a powerful sclerotic and cytotoxic agent that allows small-caliber arterial feeders to be obliterated. Its capacity to devascularize is potent. It causes anoxic cell damage, protein precipitation, and fibrinoid necrosis of the intimal lining [30]. Because its viscosity is low, alcohol can permeate more distally than other agents and cause sclerosis from within the tumor. The use of ethyl alcohol, however, is associated with a high risk of cranial nerve deficits or normal tissue infarction [3,22]. It also induces a robust inflammatory response that obliterates tissue planes and is directly toxic to the parent vessel and target tissue. Horowitz et al [31] embolized a carotid body tumor with ethanol in conjunction with distal balloon occlusion. They circumvented the limitations mentioned previously by reducing the dosing rate (4×10^{-5} mL/kg/s) and resecting the lesions within 24 hours of embolization. During surgery, alcohol injected intratumorally has yielded devascularization. Before alcohol is injected, aspiration is necessary to verify that a large vessel is not receiving this sclerotic agent. Lonser et al [32] treated three spinal epidural

masses and one posterior fossa hemangioblastoma with direct intratumoral injection of ethanol. They avoided exposing normal tissues and used a small needle (28-gauge) to introduce the embolisate into the tumor. The end point was visible blanching of the tumor. They recommended intratumoral injection of ethanol as an inexpensive universally available method of augmenting preoperative embolization. Ethanol has proven to be a powerful embolisate with a well-defined, albeit limited, role.

Coils

The primary utility of coils in tumor embolization is their ability to augment or facilitate the effects of other embolisates. Liquid or fibered coils are most commonly used for tumor embolization. Liquid coils are soft and injectable. Fibered coils are pushed mechanically through the microcatheter with a coil pusher. Coils may be used to obliterate potentially dangerous EC-IC connections before embolization with particulates. After particulate embolization, they also can be used to obliterate the proximal aspect of a feeding artery to reduce the rate of recanalization.

Gelatin foam

Gelatin foam particles are typically 40 to 60 μm; therefore, their use achieves deep tissue penetration with subsequent necrosis. Gelfoam has the advantage of being easy to use; it is easily delivered through a microcatheter without friction or blockage. It is quickly degraded by native proteolytic mechanisms, however. Embolized vessels thus recanalize fairly rapidly. Furthermore, their small caliber increases the risks of embolization of the vaso vasorum of the cranial nerves and of unpredictable embolization via EC-IC collaterals. As with other embolisates, there is a learning curve in avoiding these complications.

Fibrin glue

Fibrin glue has been used successfully to embolize tumors. The radiopacity of the fibrin glue enables continuous monitoring during embolization. Probst et al [14] argue that fibrin allows the most distal loading of the vascular bed and decreases the potential for reflux. In their series of 80 patients in whom fibrin glue was used for embolization, 2 patients suffered permanent neurologic deficits (hypesthesia in the trigeminal nerve distribution and incomplete facial paresis).

Microfibrillar collagen

Microfibrillar collagen is prepared from purified bovine collagen. It is frequently used as a topical agent during surgery and effectively controls capillary hemorrhage. It promotes platelet aggregation and is effective even in the presence of underlying coagulopathies or heparinization. Kumar et al [21] contend that the semiliquid suspension readily passes through microcatheters and that the collagen can penetrate end arteries more effectively than PVA.

Phenytoin

Phenytoin is rarely used as an embolisate. Kasuya et al [33] demonstrated that phenytoin administered via a microcatheter at a dose of 250 to 500 mg resulted in ischemic and hemorrhagic necrosis with devascularization of meningiomas. They suggested that phenytoin has the added advantages of producing precapillary microthrombosis and more complete devascularization than other agents. They recommended the perioperative administration of steroids as prophylaxis against malignant edema.

Miscellaneous

Kubo et al [34] used hydroxyapatite ceramic microparticles to embolize meningiomas in 13 patients. They noted excellent biocompatibility, good injection control, and excellent occlusion of the distal capillary bed. No microcatheters became clogged. Histologic analysis revealed mild inflammation with lymphocytic infiltration. No patient suffered a hemorrhage after embolization.

Complications of embolization

Minor and major complications can be associated with preoperative embolization. Table 1 illustrates the complications that we have noted in patients treated since 1995 at our institution. The most common complications of embolization are fever and localized pain [35]. Potentially more devastating complications include inadvertent delivery of embolisate into the intracranial circulation, intracranial or intratumoral hemorrhage, and cranial nerve injury.

Embolization of ECA branches can be complicated by unrecognized collateral connections with the posterior circulation or ICA, resulting in delivery of embolisate into the intracranial circulation. EC-IC collaterals that warrant particular note include the anastomosis between the

Table 1
Complications of tumor embolization (Barrow Neurological Institute series: n = 52)

Complication	Number (%)
Death	0 (0)
Stroke	0 (0)
Postoperative fever	1 (2)
Arterial perforation (extracranial)	1 (2)
Cranial nerve deficits	(4)
Transient	2
Permanent	0
IC embolization through EC-IC collaterals (asymptomatic)	1 (2)

Abbreviations: EC, indicates extracranial; IC, intracranial.

ophthalmic artery and the meningolacrimal branch of the middle meningeal artery. Branches of the middle meningeal and accessory meningeal arteries can have reciprocal communications with branches of the cavernous segment of the ICA. The vertebral artery shares collaterals with the ascending pharyngeal artery and the occipital artery at the odontoid arterial arch and the interspaces of C1 to C2, respectively. The internal maxillary artery may have reciprocal connections with branches of the cavernous ICA, namely, between the artery of the foramen rotundum and the anterolateral branch of the inferolateral trunk, between the accessory meningeal artery and the posteromedial branch of the inferolateral trunk, and between the middle meningeal artery and the posterolateral branch of the inferolateral trunk.

In the presence of an unrecognized patent foramen ovale, Horowitz et al [36] showed that particulate embolization may result in paradoxic embolization and subsequent stroke. They recommended the liberal use of neurophysiologic monitoring in patients undergoing general anesthesia and frequent neurologic examinations in sedated patients. Intraoperative monitoring of somatosensory evoked potentials has increased the safety of embolization by enabling the early identification of ischemia [37].

Embolization can injure cranial nerves by interrupting the vascular supply of the cranial nerves (vaso vasorum), which is often derived from the ECA. A petrous branch of the middle meningeal artery may supply the facial nerve, and the neuromeningeal branch of the ascending pharyngeal artery frequently supplies the spinal accessory and hypoglossal nerves. The potential blood supply of a cranial nerve cannot readily be identified from the dynamic anatomic information provided by angiography; therefore, provocative testing with lidocaine has been used to determine if a potentially embolizable branch supplies a cranial nerve. Horton and Kerber [38] described 26 patients who underwent the injection of 2% lidocaine mixed in equal volumes with Conray 60 (Mallinckrodt, St. Louis, Missouri). The injection (30–70 mL) was monitored with continuous fluoroscopy to ensure that no reflux occurred into the ICA. Continuous cardiac monitoring was also performed. Patients with heart block did not undergo this lidocaine challenge. Patients underwent embolization with PVA and Gelfoam particles if cranial nerve function remained stable. If the lidocaine test result was positive, the catheter was removed from the vessel and the palsy was allowed to resolve. The use of provocative testing with lidocaine for potentially embolizable intracranial branches is not widespread, because the rates of false-positive and false-negative results are high and because reflux of lidocaine intracranially can cause seizures.

Applications of preoperative embolization

The principles of embolization, choice of embolic agents, and potential complications associated with commonly embolized tumors, namely, meningiomas, hemangiopericytomas, hemangioblastomas, paragangliomas, and juvenile nasopharyngeal angiofibromas, are discussed next. An abbreviated list of hypervascular CNS tumors is given in Box 1. The variety of hypervascular tumors embolized at our institution is demonstrated in Table 2, the Barrow Neurological Institute experience since 1995.

Meningiomas

Meningiomas are typically benign and potentially curable tumors. The cell of origin is the arachnoid cap cell. They constitute 13% to 18% of intracranial tumors and have a female predominance [39]. Rates of tumor recurrence have been estimated at 9% to 11% if the dural attachment is excised, at 19% to 22% if the dural attachment is left in place, and at almost 40% if the tumor is excised subtotally [40–43]. Incomplete tumor removal may be related to tumoral hemorrhage during surgery, particularly with hypervascular meningiomas. Angiography allows the arterial supply to the tumor to be determined. It shows the site of dural attachment; the presence

Box 1. Hypervascular central nervous system tumors

Intra-axial
Hemangioblastomas
Metastatic tumors
Glioblastomas multiforme

Extra-axial
Meningiomas
Hemangiopericytomas
Juvenile nasopharyngeal angiofibromas
Paragangliomas
Schwannomas
Hemangioendotheliomas

Skull tumors
Aneurysmal bone cysts
Hemangiomas
Ewing's sarcomas
Mesenchymal chondrosarcomas

of displacement; or the degree of encasement of key vascular structures, including the dural venous sinuses. It also helps to determine the vascularity of a tumor.

The vascular supply of meningiomas is twofold. Arterial feeders to the pedicle at the site of attachment and center of the tumor typically arise from branches of the ECA and supply the tumor radially to produce the characteristic "sunburst" appearance on angiography. The apex of the sunburst is usually the site of dural attachment. In most cases, pial and cortical arteries supply the capsule, and this contribution increases as the tumor enlarges. Dural pedicle feeders from the ECA include the middle meningeal artery, accessory meningeal artery, neuromeningeal branch of the ascending pharyngeal artery, and stylomastoid branch of the occipital artery. The dural supply from the ICA is usually from the ethmoidal, cavernous, clival, or tentorial branches. Depending on the location of the meningioma, the primary supply to the meningioma may be from the ICA, ECA, or both. Meningiomas supplied solely by the ICA include diaphragmatic or tuberculum sellar lesions.

Anterior fossa lesions are supplied by the ECA and ICA. High-convexity and parasagittal lesions are supplied by the middle meningeal artery and the artery of the falx cerebri. All parasagittal lesions must be examined for contributions from the contralateral middle meningeal artery. Frontal convexity or frontal falcine tumors are supplied by a combination of the meningeal branches of the ethmoidal artery and anterior falcine branches. Bilateral anterior and posterior ethmoidal arteries usually supply olfactory groove meningiomas. As a result, the distal internal maxillary branches and the middle meningeal artery must be evaluated.

Middle fossa tumors are supplied by branches from the ECA, including the artery of the foramen rotundum, vidian arteries, and ascending pharyngeal artery. In particular, meningiomas involving the sphenoid wing are supplied by the recurrent meningeal branch of the ophthalmic artery or branches of the middle meningeal artery. Parasellar tumors are frequently fed by branches of the petrous, cavernous, and supraclinoid segments of the ICA; the artery of the foramen rotundum; the artery of the foramen ovale; and the neuromeningeal branch of the ascending pharyngeal artery.

Table 2
Barrow Neurological Institute series of embolized central nervous system tumors (1995–present)

Tumors	Number
Meningiomas	
Olfactory groove	1
Parasagittal	2
Tentorial	2
Convexity	5
Sphenoid wing	1
Atypical	1
Juvenile nasopharyngeal angiofibromas	11
Hemangioblastomas	4
Paragangliomas	
Glomus tympanicum	1
Glomus jugulare	7
Glomus vagale	1
Carotid body tumor	1
Hemangiopericytomas	1
Others (14)	
Angiosarcoma	1
Vestibular schwannoma	1
12^{th} nerve schwannoma	1
Ewing sarcoma	1
Hemangioma	1
Metastatic renal cell (cranial)	1
Aneurysmal bone cyst (spinal)	1
Plasmacytoma (spinal)	2
Giant cell tumor (spinal)	1
Spinal metastasis	3
Schwannoma (spine)	1

Posterior fossa meningiomas are primarily supplied by the posterior meningeal artery, the middle meningeal artery, and the accessory meningeal artery. The tentorial branch of the meningohypophyseal trunk, the inferolateral trunk, the middle meningeal artery, and the accessory meningeal artery can all supply tentorial meningiomas. These branches also may supply the third through sixth cranial nerves. Consequently, provocative testing may be beneficial. Petroclival meningiomas are supplied by the petrosal, petrosquamosal, and occipital branches of the middle meningeal artery; the transmastoid branches of the occipital and posterior auricular arteries; the subarcuate branch of the anterior inferior cerebellar artery (AICA); and neuromeningeal branches of the ascending pharyngeal artery. The posterior meningeal artery arising from the vertebral artery or branches of the ascending pharyngeal artery [1] supply meningiomas involving the foramen magnum. Posterior fossa tumors share their blood supply with the lower cranial nerves. This point must be recalled when embolizing these tumors. Also, EC-IC anastomoses, specifically between branches of the posterior auricular or occipital artery and the vertebral artery in the high cervical spinolaminar region, may be present.

Angiography is instrumental in determining the patency of the major dural venous sinuses. Meningiomas can invade and occlude a major sinus. Preoperative magnetic resonance venography or angiography can verify invasion, occlusion, and collateral venous drainage and thereby facilitate surgical decision making. If gross total resection is the goal, preexisting occlusion of a sinus allows more aggressive tumor resection by opening the sinus or resecting the involved segment.

In 1973, Manelfe et al [44] first described the preoperative embolization of meningiomas. Transcatheter embolization has been advocated to reduce the vascularity of tumors, to facilitate necrosis of the dural attachment site, to mitigate tumoral hemorrhage, and to facilitate tumor resection [2,19,45,46].

The most commonly used embolisate for meningiomas is PVA. Particles in the range of 150 to 350 μm are preferred because they can penetrate deeply into the tumor substance. If reflux into the parent artery occurs, embolization should be discontinued. Ideally, the postembolization angiographic goal is obliteration of the tumor blush on injection of the ECA. If embolization obliterates the feeding arteries but the tumor blush remains, the tumor is likely to remain hypervascular.

The presence of estrogen and progesterone receptors in many meningiomas introduces the attractive concept of ligand-specific selectivity to preoperative embolization. A report by Suzuki and Komatsu [47] using estrogen to embolize dural AVMs and meningiomas suggests that estrogen or progestins may be used as embolisates for tumors. Although the mechanism of action is unclear, estrogen is thought to injure the vascular endothelium and to increase vascular permeability.

Most meningiomas do not require preoperative embolization because the tumor can be devascularized during surgical resection as a first step. Embolization is valuable for large hypervascular skull base meningiomas with an arterial supply that is not readily accessible surgically [48]. Embolization should be considered for giant meningiomas (Fig. 1), meningiomas involving the skull base and middle cranial fossa, falcine or parasagittal meningiomas, and meningiomas in the pineal region. In patients with skull base meningiomas, the vascular pedicle is seldom encountered until a significant portion of the tumor has been resected, making embolization more imperative. To forestall tumor progression, embolization may be considered for patients who are poor surgical candidates.

Embolization of deep arterial feeders, such as the meningohypophyseal trunk and inferolateral trunk, is technically challenging, because their caliber is small and their angle of origin is acute. The introduction of variable stiffness and hydrophilic microcatheters and microwires has permitted selective microcatheterization of these vessels, however. Hirohata et al [49] described seven patients with large petroclival meningiomas who underwent preoperative embolization with 150- to 250-μm particulates. Branches of the meningohypophyseal trunk and inferolateral trunk (lateral clival, posterior branch, and tentorial branch), which provided the primary blood supply, were catheterized successfully. They did not use lidocaine because of the high reported rates of false-positive and false-negative results and for fear of introducing an epileptogenic agent into the distal territory [50,51]. All tumors were subtotally or completely resected; blood loss was 500 mL or less. Robinson et al [51] described five patients with skull base meningiomas who underwent successful preoperative embolization of the

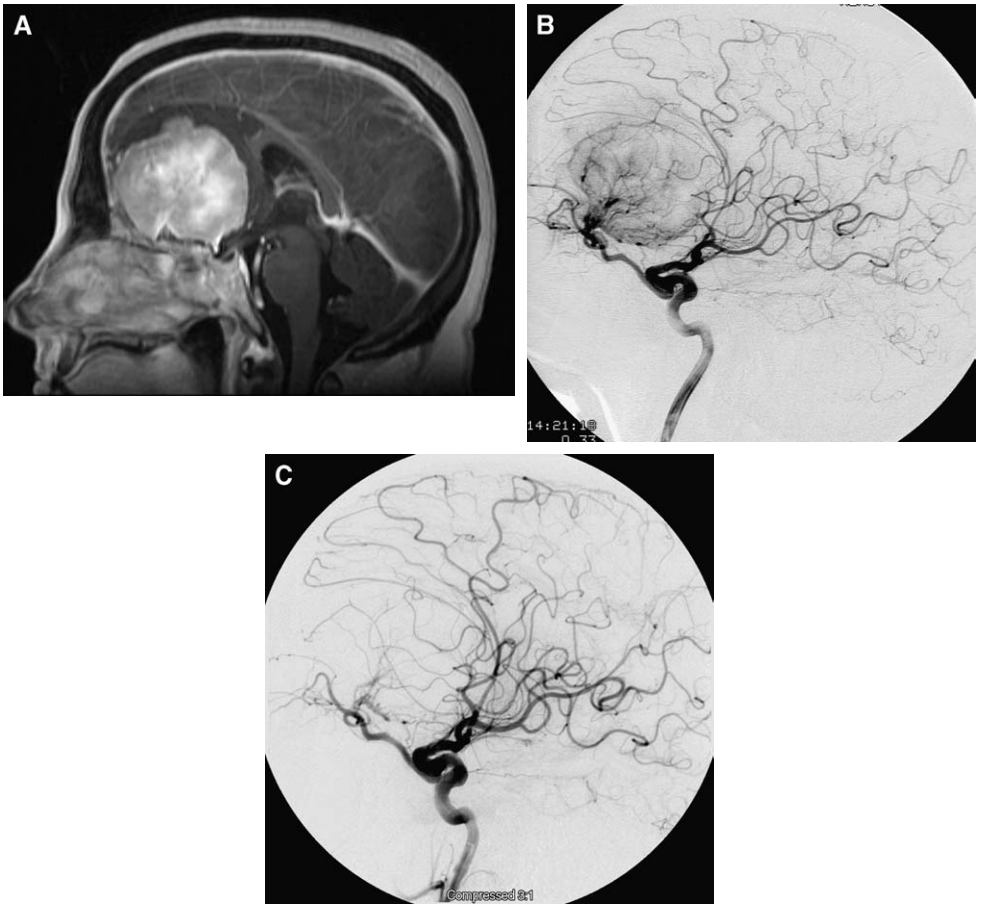

Fig. 1. (*A*) Sagittal MRI shows a giant anterior skull base meningioma. (*B*) Lateral internal carotid artery angiographic injection shows the "sunburst" pattern of arterial supply. Ethmoidal branches arising from the ophthalmic artery feed this giant tumor. (*C*) The vascularity is markedly reduced after glue embolization.

meningohypophyseal trunk and inferolateral trunk with particulate embolisates. No patient suffered complications, and the tumor blush was obliterated in 80% to 100% of the cases. The inferolateral trunk, however, must be embolized with caution because it can collateralize with the ophthalmic artery via the deep recurrent ophthalmic artery. Embolization of the inferolateral trunk is thus associated with the risk of blindness.

Pial and ophthalmic arteries supplying meningiomas are difficult to embolize, and the risk-benefit ratio often precludes an attempt. Kaji et al [52] described two cases in which distal cortical branches of the ICA were successfully and safely embolized with Gelfoam before surgical resection. They emphasized that embolization of the pial supply should be attempted only under the following conditions: the tumor is supplied solely by the ICA, the tumor is located in a noneloquent region of the brain, the patient has a negative Amytal test result, superselective microcatheterization is performed with the catheter abutting the tumor, and particles are used rather than acrylic glue.

Pineal region meningiomas are typically supplied by meningeal branches of the ECA, the tentorial artery, the medial and lateral posterior choroidal branches, the posterior pericallosal artery, small branches of the posterior cerebral artery, and branches of the superior vermian and superior cerebellar arteries (SCAs). Meningeal branches may arise from the vertebral artery and PICAs. Sagoh et al [53] successfully resected a pineal region meningioma after embolizing

bilateral middle meningeal arteries with estrogen-alcohol and PVA. Postembolization MRI showed intratumoral necrosis.

Optic nerve meningiomas are seldom amenable to embolization because they are supplied by branches of the ophthalmic artery that concurrently supply the nerve. Terada et al [54] embolized five hypervascular meningiomas fed primarily by branches of the ophthalmic artery. One patient was blind before embolization, and another patient suffered a visual field deficit after embolization. They contended that this technique is feasible if the microcatheter is distal to the origin of the central retinal artery and emphasized that reflux into this artery must be avoided.

Bendszus et al [55] prospectively studied the effects of preoperative embolization on the excision of meningiomas at two similar centers. Thirty patients each were enrolled in the embolization and nonembolized groups. One patient in the embolization group suffered a permanent complication (thromboembolic occlusion of the central retinal artery). Overall, there was no significant difference in intraoperative blood loss. Only the subgroup of patients who underwent complete embolization without residual tumor blush had significantly less intraoperative blood loss. These investigators concluded that the value of nonselective preoperative embolization of meningiomas may be limited, especially given the time, expense, and complications associated with embolization.

A retrospective study from our institution examined the utility and risk-benefit profile of 33 appropriately matched embolized and 193 nonembolized meningiomas. Costs of treatment for the two groups were also compared. Preoperative embolization significantly reduced the intraoperative blood loss and need for transfusions associated with large meningiomas. There were no differences between the two groups in terms of cost, length of hospital stay, and rates of major or minor complications. Therefore, embolization may be beneficial for large meningiomas.

Bendszus et al [56] prospectively followed seven patients who underwent embolization with trisacryl gelatin microspheres (100–300 μm) alone without surgery. At a mean radiographic follow-up of 20 months, the tumors of six of the seven patients were smaller than before treatment. The reduction was most pronounced 6 months after treatment. They contended that embolization alone may be an option for patients who are poor surgical candidates. The drawback of this treatment modality is the lack of histologic verification of the diagnosis. Long-term follow-up is also required to determine the efficacy of treatment.

Complications

The overall risk associated with the embolization of meningiomas is low [19,57]. Minor complications include painful trismus, facial pain, or both and may occur in as many as 20% to 30% of patients [19]. Treatment of these complications includes corticosteroids and analgesic medications. In most patients, symptoms resolve within 2 to 3 days.

Major complications include stroke, blindness, hemorrhage, and cranial nerve palsies. Stroke or blindness is rare but can be the result of unappreciated EC-IC collaterals or can be caused by reflux of embolic material. These complications can be avoided by thorough angiographic evaluation, including a superselective examination to delineate anastomoses between the meningeal vessels and the ICA, vertebral artery, or ophthalmic artery.

Seventh cranial nerve palsy results from inadvertent embolization of the petrous branches of the middle meningeal artery, which supplies posterior parasellar and posterior fossa lesions. Lower cranial nerves are at risk when clival or petroclival meningiomas supplied by branches of the ascending pharyngeal artery are to be embolized. The risk of cranial nerve palsies can be mitigated by superselective catheterization of the external branches until the catheter tip is wedged within the vessel supplying the tumor. Cranial nerve damage is also less likely with particle embolization, specifically when larger particulates are used. Using particulates larger than 150 μm is thought to prevent inadvertent embolization of the vaso vasorum supplying the cranial nerves. Probst et al [14] reported cranial nerve deficits in 2 of 80 patients undergoing embolization, which were temporary in 1 patient and permanent in the other.

Hieshima et al [58] reported no permanent neurologic complications in 11 patients undergoing embolization for a meningioma. Richter and Schachenmayr [2] described 5 patients with transient neurologic deficits and no permanent deficits in 31 patients whose meningiomas were embolized. In a series of 51 patients who underwent embolization, Macpherson [46] described 8 patients who experienced scalp necrosis or

temporary hemiparesis. Rosen et al [59] embolized skull base meningiomas in 167 patients, an ostensibly high-risk group for embolization or surgery. Transient neurologic deficit occurred in 12.6%, and 9% had permanent neurologic deficits. In their experience, embolization of the meningohypophyseal trunk, ascending pharyngeal artery, and middle meningeal artery was associated with a high risk of transient and permanent neurologic deficits. Swelling after embolization was readily controlled with intravenous steroids. If swelling persists, emergent surgical resection must be considered. Patients with skull base meningiomas frequently have baseline cranial nerve dysfunction. In this subgroup of patients, embolization may exacerbate this dysfunction, and this possibility should be highlighted during preoperative counseling [59]. Two patients developed monocular blindness after embolization, and neither patient was embolized via the ophthalmic artery.

Subarachnoid, subdural, peritumoral, or intratumoral hemorrhages have followed the embolization of meningiomas [60–64]. Hemorrhage may be caused by wire perforation or sudden dynamic changes in intracranial blood flow as a result of the embolization [60]. After reviewing the literature on the postembolization risks of intratumoral hemorrhage, Kallmes et al [65] were unable to discern a correlation between particle size and risk of hemorrhage. They found seven cases of hemorrhage into a meningioma. Eliminating the supply from the ECA to tumors with a significant ICA supply has been reported to increase blood flow from the ICA, exacerbating mass effect and intratumoral hemorrhage [48,66]. This potential complication must be considered when tumors have a significant ICA supply. Intratumoral hemorrhage also may be more common in tumors with cystic components or large necrotic areas than in homogeneous lesions [64]. Barr et al [67] described an iatrogenic carotid-cavernous sinus fistula that presumably resulted from microwire perforation of a meningohypophyseal artery supplying a skull base meningioma. The fistula was treated with transarterial coil embolization of the venous pouch. Given the risk profile, they concluded that skull base meningiomas must not be embolized indiscriminately.

Scalp necrosis can also follow embolization and surgical resection. Adler et al [68] described severe scalp necrosis treated with a vascularized free tissue transfer. Chan and Thompson [69] and Adler et al [68] emphasized the need to base the scalp flap on at least one patent supplying artery to prevent this complication. They also recommended maintaining the superficial temporal artery as a potential donor vessel for a free tissue transfer. Scalp necrosis is rare when larger particulates are used as the embolisate.

A potential concern is that embolized meningiomas may be overgraded on histologic examination because of the embolization-induced necrosis and reactive changes [69,70]. Ng et al [70] suggested that embolization-induced necrosis is characterized by a punched-out outline with confluent areas of necrosis. Embolized meningiomas lack the overall background of anaplasia associated with atypical or aggressive meningiomas. Perry et al [71] examined 64 embolized meningiomas and concluded that these morphologic changes are uncommon and that current grading schemes rarely overgrade these tumors. They found a higher proportion of atypical meningiomas in patients undergoing embolization but attributed it to selection bias rather than to the effects of embolization.

Hemangioblastomas

Hemangioblastomas constitute 1.1% to 2% of craniospinal tumors [72]. They most frequently occur within the cerebellar hemispheres and rarely at the vermis, cerebellopontine angle, or brain stem. Although most of these lesions are sporadic, approximately 20% are associated with von Hippel-Lindau (VHL) disease. VHL disease is transmitted in an autosomal dominant fashion with incomplete penetrance. Multiple hemangioblastomas are the norm in patients with this disease. The tumors are hypervascular, which makes their resection exceedingly difficult, particularly in eloquent areas. Severe intraoperative hemorrhage is a significant contributor to the morbidity and mortality rates associated with the resection of hemangioblastomas [73]. Before the advent of microsurgical techniques, morbidity and mortality rates approached 50% [74].

On angiography, the blood supply is typically via the PICA and, less commonly, via the AICA or branches of the SCA branches. Pontomedullary hemangioblastomas can recruit supply from the SCA. Branches of the vertebral artery or anterior spinal artery may supply cervicomedullary lesions. Dural branches of the vertebral artery, such as the posterior meningeal artery, may supply superficial lesions. The caliber of

feeding arteries can exceed that of the basilar artery.

The criteria for embolizing hemangioblastomas include large tumors with well-defined arterial feeders that are not readily accessible surgically (Fig. 2). Preoperative embolization has been advocated for lesions larger than 3 cm [75]. The risk of embolization is high with hemangioblastomas, because the feeding arteries are often pial vessels (ie, branches of the PICA or AICA). Embolization should be performed with the microcatheter tip placed beyond the normal branches. The embolisate of choice is PVA or NBCA.

Tampieri et al [76] treated two patients with large hemangioblastomas, one spinal and one involving the posterior fossa, with preoperative embolization. Both lesions were then resected with blood loss of less than 100 mL. Eskridge et al [77] treated nine patients with craniospinal hemangioblastomas with PVA embolisate and incurred no permanent complications. One patient developed malignant posterior fossa edema associated with hydrocephalus after treatment, however. They advocated perioperative steroids, intensive care unit observation, and surgical resection within 48 to 72 hours because of the potential for recanalization of feeding arteries after embolization with PVA. The surgeons believed that embolization facilitated tumor manipulation and surgical resection.

Conway et al [78] described 4 of 40 patients with hemangioblastomas who underwent preoperative embolization. In a patient with a sacral

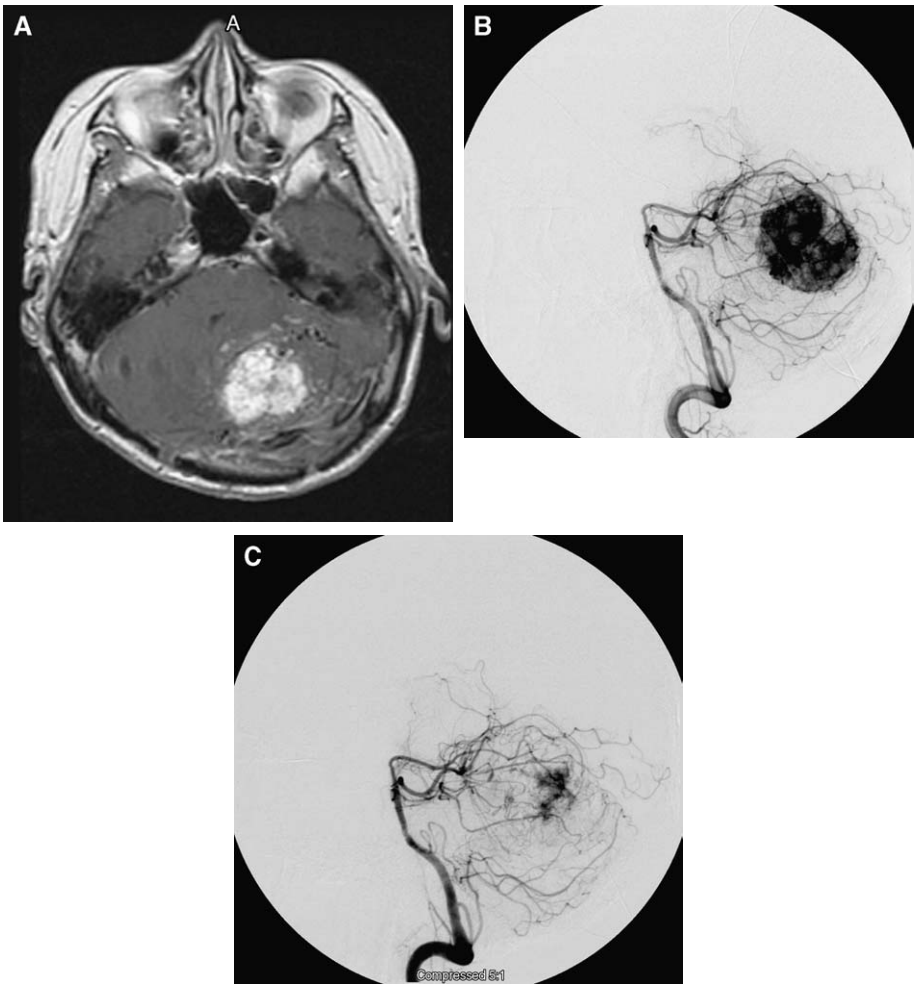

Fig. 2. (*A*) Axial postcontrast MRI shows an intensely enhancing posterior fossa mass. (*B*) The vertebral artery injection shows the dense vascularity of a hemangioblastoma. (*C*) The deep arterial supply is obliterated with glue embolization.

hemangioblastoma, embolization alone was sufficient to arrest progression of the patient's symptoms. One patient suffered a lateral medullary infarction after embolization of a medullary hemangioblastoma. These investigators recommended reserving embolization for tumors with large surgically inaccessible feeders.

Lee et al [79] treated 4 of 14 patients with spinal cord hemangioblastomas with embolization and surgical resection. Complete resection was achieved in all four cases. Subjectively, the surgeon reported significantly less blood loss in the embolized patients. Contraindications to embolization included supply to the hemangioblastoma from the artery of Adamkiewicz and a surgically accessible arterial supply. Four patients were treated before the advent of spinal embolization techniques. Tumor feeders emanating from the anterior spinal axis should not be embolized because of the risk of occluding the anterior spinal artery.

Hemangiopericytomas

Meningeal hemangiopericytomas are aggressive tumors originating from the contractile pericytes of Zimmerman, which envelop capillaries. They are rare, accounting for less than 1% of all CNS tumors [80]. They occur equally in both genders and are an affliction of middle age. They are associated with a high risk for recurrence and carry metastatic potential [81].

These tumors are quite vascular, and intraoperative bleeding can be significant. Hemorrhage is the most common cause of surgical morbidity and mortality as well as the primary reason for subtotal resection [78,90]. Embolization of these tumors must be aggressive, and the use of ethanol as well as direct surgical puncture should be considered (Fig. 3).

Several reports have described the utility of embolization in mitigating surgical blood loss [82–84]. Embolization of these tumors is exceptionally difficult because they parasitize the cortical vessels [79,81]. Reported complications include permanent Horner's syndrome [82]. Embolization alone typically provides insufficient tumor control or cure.

Muraszko et al [85] treated four patients with hemangiopericytomas of the spine with embolization and surgical resection. They advocated embolization to reduce the vascularity of the tumor. They caution that preoperative deficits may worsen, likely because the tumor swells after embolization. They therefore recommend urgent surgical resection after embolization.

Paragangliomas

Paragangliomas are typically benign slow-growing neoplasms that arise from neural crest paraganglion cells. In the head and neck, they occur in the temporal bone (glomus tympanicum and glomus jugulare; Fig. 4), the carotid bifurcation (glomus caroticum), and the upper parapharyngeal space (glomus vagale; Fig. 5). On extremely rare occasions, paragangliomas can be found within the spinal canal [86].

Approximately 4% of paragangliomas have documented catecholamine secretion [87–90]. This behavior can cause a pheochromocytoma-type syndrome associated with broad fluctuations in blood pressure or marked hypertension. If catecholamine secretion is documented, the patient must be pretreated with α- and β-blockade before embolization and surgical resection. The most common presenting symptoms include progressive unilateral hearing loss and pulsatile tinnitus.

Angiography must define the intracranial and extracranial arterial supply to the tumor as well as the involvement of the dural venous sinus. The patency of both transverse-sigmoid systems must be evaluated to determine if the involved sinus can be sacrificed without causing intracranial venous hypertension. The primary blood supply is from branches of the ascending pharyngeal artery. Temporal glomus tumors often recruit their blood supply from petrous branches of the ICA (eg, vidian artery) and from cavernous-carotid branches (clival branch of the meningohypophyseal trunk). Glomus tympanicum are usually small lesions that can be resected with conventional tympanoplasty techniques without preoperative embolization.

Glomus jugulare lesions, particularly those with an intracranial extension, require preoperative embolization [91]. Preoperative embolization of these tumors helps to reduce intraoperative blood loss, increases the extent of tumor resection, and decreases the length of surgery [92–95]. Reduced intraoperative bleeding permits more meticulous dissection of the crucial neurovascular structures at the skull base. Most glomus jugulare tumors tend to have a multicompartmentalized arterial supply. For embolization to be effective, each compartment should be catheterized selectively and embolized. The inferomedial compartment is supplied by the ascending pharyngeal

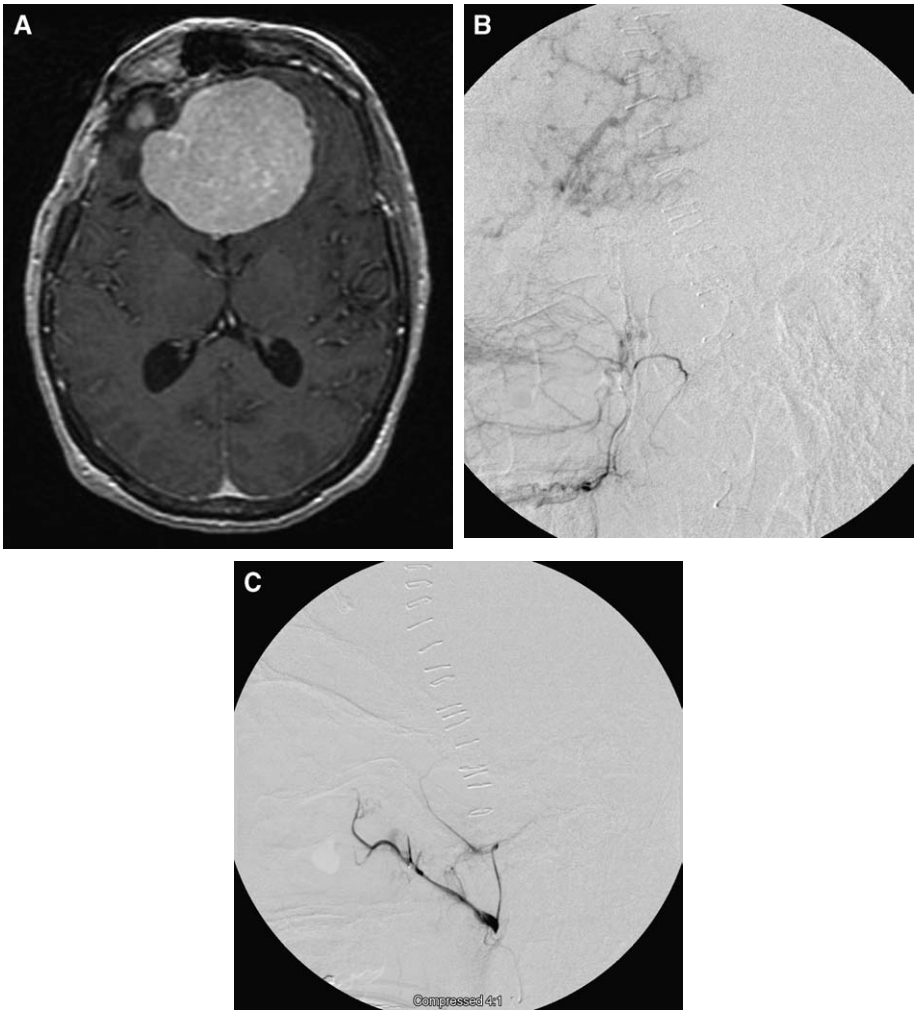

Fig. 3. (*A*) Axial postcontrast T1-weighted MRI shows a giant anterior skull base hemangiopericytoma. (*B*) Superselective injection of the internal maxillary artery shows a prominent tumor blush. (*C*) No tumor blush is seen after embolization with particulates (polyvinyl alcohol).

artery. The posterolateral compartment is supplied by the stylomastoid branch of the occipital or posterior auricular artery. The anterior compartment is supplied by branches of the internal maxillary artery and ICA (caroticotympanic artery), and the superior compartment is supplied by branches of the middle meningeal artery. Superselective microcatheterization can help to define this anatomy and to identify EC-IC anastomoses [96]. For tumors with significant supply from the ICA and those that encase the ICA, balloon test occlusion with preoperative sacrifice of the ICA is an option. Cohen et al [97] reported a case in which the tumor was devascularized and the ICA was preserved by using a covered stent placed within the petrous ICA.

Carotid body tumors occur sporadically, and bilateral tumors occur in 5% of cases. These lesions typically manifest in the fourth to sixth decades of life as a painless enlarging neck mass. Local mass effect, however, can cause dysphagia, hoarseness, stridor, tongue paresis, and vertigo. Continued growth leads to involvement of the ICA, ECA, and vagus and hypoglossal nerves. Pharyngeal compression with skull base and intracranial extension also occurs. Malignancy, as defined by distant metastatic spread, occurs in 2% to 6% of lesions [98–100]. Surgical excision can be

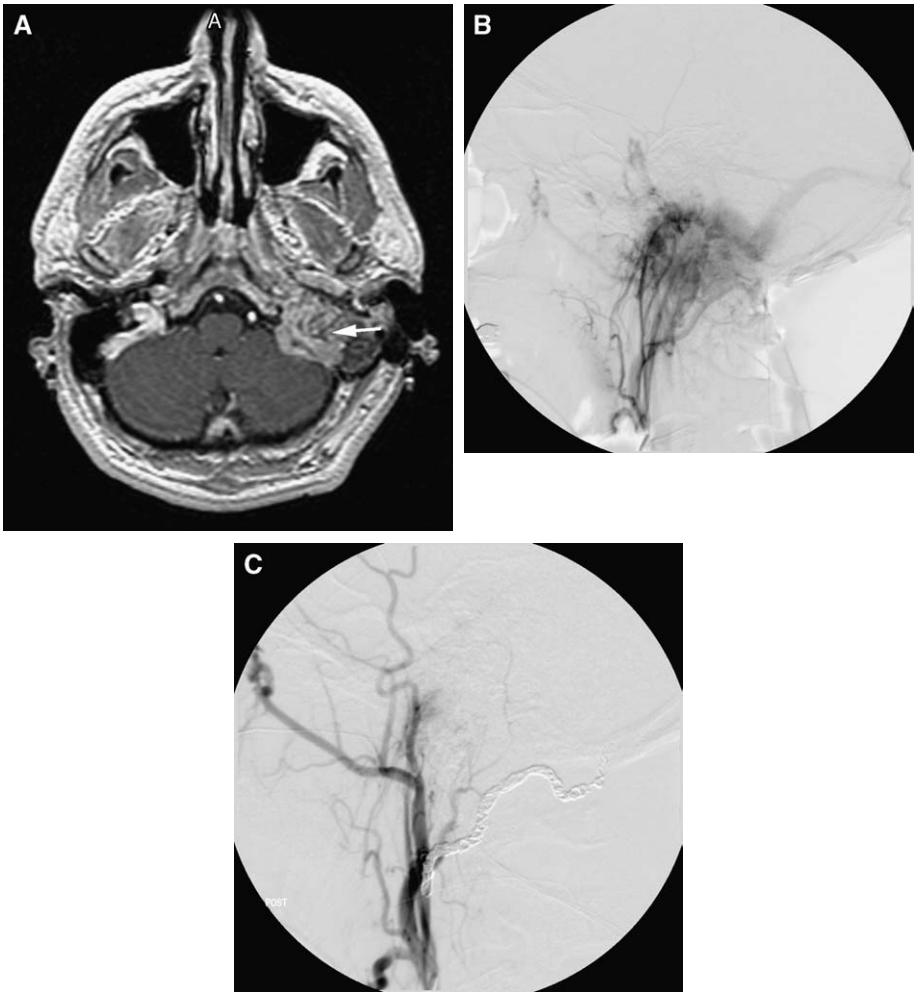

Fig. 4. (*A*) Axial postcontrast MRI shows an extensive left glomus jugulare tumor (*arrow*). (*B*) This tumor has a profound vascular supply derived from multiple branches of the external carotid artery. (*C*) The vascularity of the tumor is reduced drastically after glue and coil embolization.

challenging, because the tumors are hypervascular and they can involve the cranial nerves extensively.

LaMuraglia et al [101] embolized 11 patients with carotid body tumors before surgery. Tumors were supplied via the ascending pharyngeal artery, ascending cervical branches of the thyrocervical trunk, and vertebral artery. One patient suffered transient aphasia that resolved within 24 hours. Embolization significantly decreased intraoperative bleeding compared with nonembolized lesions. These investigators recommended preoperative embolization for tumors larger than 3 cm, followed by surgical resection within a few days. Embolization can be difficult because of the tumor's location at the carotid bifurcation. Therefore, they advocated temporary balloon occlusion with hypotensive challenge if the carotid artery is extensively involved. Borges et al [102] described 2 patients whose large carotid body tumors were completely resected with minimal blood loss after preoperative embolization with PVA.

Complications

Similar to embolization of other intracranial tumors, most severe complications are related to delivery of the embolisate into the intracranial circulation via reflux or through EC-IC collaterals. Palsy of the lower cranial nerves (IX–XII) can

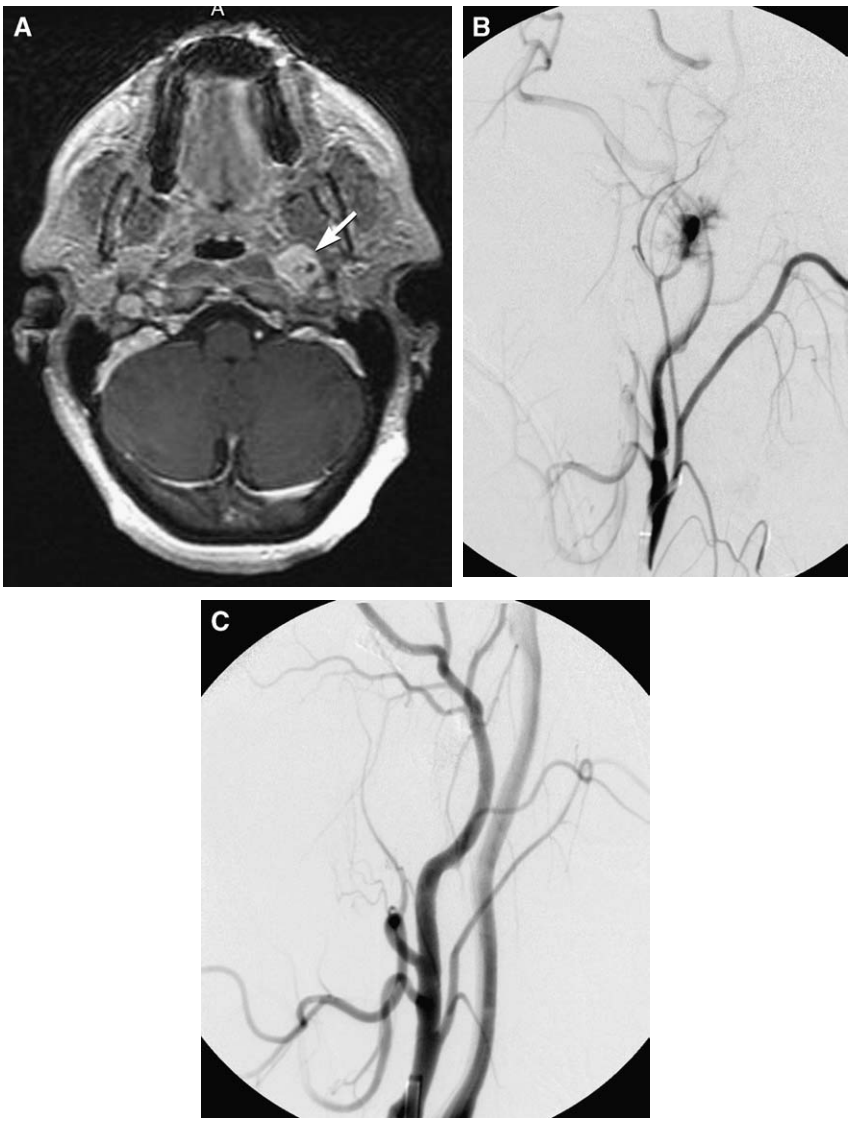

Fig. 5. (*A*) Axial postcontrast MRI shows an enhancing tumor consistent with a glomus vagale in the left carotid sheath (*arrow*). (*B*) The predominant blood supply is from a branch of the ascending pharyngeal artery. (*C*) No residual supply to the tumor is seen after glue embolization.

follow embolization of the vaso vasorum supplying these nerves. In an early report, Pandya et al [103] described a herniation syndrome that followed direct puncture and Gelfoam embolization of a glomus jugulare tumor. They theorized that significant tumor infarction exacerbated posterior fossa edema. Marangos and Schumacher [104] and Herdman et al [105] described two cases of facial palsy after embolization of a glomus jugulare tumor. One patient had recovered completely 1 year after treatment, and the other had improved significantly.

The facial nerve typically receives its blood supply from the stylomastoid artery and from petrosal branches of the middle meningeal or accessory meningeal artery. When the tumor has occluded the stylomastoid artery and the meningeal contribution to the nerve is small, facial nerve paresis is to be expected. Recovery of function is the rule after embolization with PVA, because the

embolized vessels eventually recanalize. When a permanent embolisate, such as glue, is used, a provocative test should be considered before proceeding.

Juvenile nasopharyngeal angiofibromas

Juvenile nasopharyngeal angiofibromas are highly vascular benign neoplasms that account for 0.5% of all head and neck neoplasms [106]. These tumors originate within the superior posterior margin of the sphenopalatine foramen. They almost exclusively afflict adolescent boys; the mean age at diagnosis is 14 years. Although benign, juvenile nasopharyngeal angiofibromas are locally invasive and exhibit high rates of recurrence after subtotal resection [107,108]. The most common presenting symptoms include nasal obstruction or epistaxis.

On nasopharyngeal examination, a pink-blue nodular mass is seen in the oropharynx. Angiography shows multiple tortuous vessels, with a dense homogeneous blush during the capillary phase. Prominent draining veins become apparent immediately. These tumors are supplied by branches of the ipsilateral internal maxillary artery (Fig. 6). The ascending pharyngeal artery is involved in as many as one third of the cases [109]. Bilateral carotid angiography is vital, because these tumors, particularly if they have an intracranial extension, often recruit their blood supply from the ophthalmic artery, contralateral internal maxillary artery, and branches of the ICA.

Preoperative embolization is considered crucial for reducing the intraoperative blood loss associated with these tumors. The aim of embolization is to occlude the small distal vessels within the tumor and not simply to obstruct the feeding arteries. In an early study, Roberson et al [109] showed that, on average, preoperative embolization reduced intraoperative blood loss from 2400 to 800 mL. Siniluoto et al [110] demonstrated significantly less blood loss, improved extent of resection, and fewer recurrences in embolized patients compared with nonembolized patients. Their sample comprised only 10 patients, however. Several other studies have reported the safety and efficacy of embolization followed by surgical resection [5,111].

Complications

Complications reported to follow embolization of juvenile nasopharyngeal angiofibromas include fever and local pain. Postembolization fever should not postpone resection. Bradycardia may follow embolization of the internal maxillary or ascending pharyngeal arteries. Intracranial embolization is usually caused by unrecognized EC-IC collaterals or reflux of embolisate. Gay et al [112] have described postoperative palatal necrosis and an oronasal fistula after staged embolization and transpalatal resection of a juvenile nasopharyngeal angiofibroma. They believe that this complication is potentiated by embolization but maintain that embolization is still warranted for these tumors.

Miscellaneous

Aneurysmal bone cysts are benign nonneoplastic lesions primarily afflicting individuals younger than 20 years of age. Although the metaphyses of long bones are the primary sites of origin, the skull is affected in 2.5% to 6% of cases [113]. CT and MRI show multiple loculations within the lesion, with peripheral sclerosis. Angiography shows a tumor blush most prominent on the outer aspect of the lesion, which is typically vascular. The core of the lesion is often avascular.

Treatment options include surgical excision or curettage, radiotherapy, cryosurgery, and embolization. The treatment of choice is curative surgical excision. Sheikh [113] reviewed the literature on the management paradigms used to treat cranial aneurysmal bone cysts. Embolization may be used to devascularize the tumor before surgery or as the only treatment modality in surgically inaccessible lesions. Ikeda et al [114] described regression of an aneurysmal bone cyst after continuous embolization with estrogen for 9 days.

Bingaman et al [115] embolized an intracranial extraskeletal mesenchymal chondrosarcoma that became symptomatic with headache, nausea, and vomiting. The lesion was located extra-axially in the right frontal region and was prominently supplied from bilateral branches of the ECA. Branches of the right middle meningeal artery were embolized. The authors stressed the importance of gross total resection and close follow-up of this potentially aggressive tumor.

Avellino et al [116] described a Masson's vegetant intravascular hemangioendothelioma involving the cerebellopontine angle and middle cranial fossa. The lesion had recurred despite embolization followed by surgical resection on two separate occasions. This exceptionally

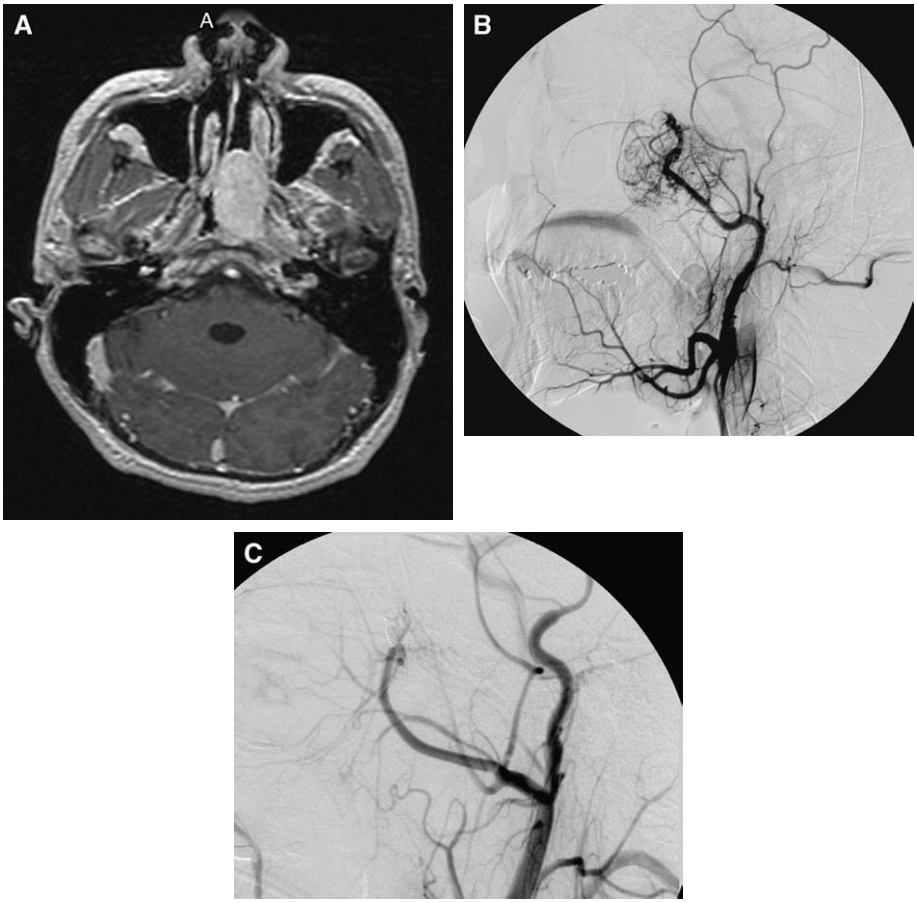

Fig. 6. (*A*) Axial MRI shows a 27-year-old man with a large juvenile nasopharyngeal angiofibroma. (*B*) The distal branches of the internal maxillary artery luxuriously supply the tumor. (*C*) Postembolization angiogram shows no residual arterial supply to this hypervascular tumor.

vascular lesion was supplied by the AICA, meningohypophyseal artery, and occipital arteries. Embolization of the left occipital artery with PVA particles at the patient's initial presentation was successful. When the tumor recurred, multiple branches of the ECA and the left meningohypophyseal artery were contributing significant blood supply. The branches of the ECA were embolized with PVA. The patient tolerated balloon test occlusion and underwent permanent balloon occlusion of the left ICA.

Summary

Preoperative embolization plays a vital role in the management of giant or skull base meningiomas and hypervascular tumors, such as hemangioblastomas, hemangiopericytomas, juvenile nasopharyngeal angiofibromas, and paragangliomas. Recent advances in microcatheter technology, microembolisates, and neurophysiologic monitoring have improved the safety of preoperative embolization. Sound endovascular principles and techniques are paramount to prevent major complications, such as stroke, blindness, or cranial neuropathy, however. Preoperative embolization of tumors has become a requisite tool for the neurosurgical team and represents a signifiant improvement in patient care.

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