Contemporary Endovascular Embolotherapy for Meningioma

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Abstract

Preoperative endovascular tumor embolization has been used for 40 years. Meningiomas are the most common benign intracranial tumor in which preoperative embolization has been most extensively described in the literature. Advocates of embolization report that it reduces operative blood-loss, and softens the tumor, thus making surgery safer and easier. Opponents suggest that it adds additional risk and cost for patients without controlled studies showing conclusive benefit. The literature suggests a 3 to 6% neurological complication rate related to embolization. The combined external and internal carotid artery blood supply and complex anastomoses of the meninges can make embolization challenging. Positive outcomes require thorough knowledge of the pertinent vascular anatomy, familiarity with the neurovascular equipment and embolics, and meticulous technique. There remains debate on several aspects of embolization, including tumors most appropriate for embolization, embolic agent of choice, ideal size of embolic, and the choice of vessel(s) to embolize. This detailed review of pertinent vascular anatomy, embolization technique, results, and complications should allow practitioners to maximize treatment outcomes in this setting.

Keywords

- meningioma
- embolization
- complications
- PVA
- Gelfoam
- interventional radiology

Objectives: Upon completion of this article, the reader will be able to discuss the role of embolization therapy in the treatment of meningiomas, including patient selection and possible complications.

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Preoperative endovascular embolization has been used in the treatment of vascular head and neck tumors for 40 years.^{1,2}

Examples of vascular tumors include meningiomas, glomus tumors, hemangioblastomas, hemangiopericytomas, nerve sheath tumors, juvenile nasal angiofibromas, and metastases. Preoperative meningioma embolization is often performed prior to surgical excision. The goal of preoperative meningioma embolization is tumoral blood flow reduction through the elimination of one (or more) arterial feeders to the tumor. Proponents of preoperative embolization argue that it reduces intraoperative blood loss, need for transfusion, overall surgical time, and makes the tumor softer and thus the procedure easier and safer.^{3–12} Some authors suggest that embolization can decrease tumor recurrence or that it may be used as a stand-alone therapy in high-risk surgical candidates.^{1,9,11–20} The procedure, however, is not without risk.^{4,8,15,21–24} Patient-specific, tumor-specific, and provider-specific factors must be weighed when contemplating the appropriateness of meningioma embolization. Every case will benefit from multidisciplinary input regarding the appropriateness of and aggressiveness with which to perform embolization. The

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lack of consistency in the literature with regard to indication, technique, and outcome reporting makes it difficult to define the exact role of preoperative meningioma embolization. This article provides the interventional radiologist (IR) a detailed knowledge of the history, technique, embolics, outcomes, and complications of this procedure.

Meningioma—General Information

Meningiomas represent the most common benign intracranial neoplasm in adults and in hospital series account for 13 to 26% of primary intracranial tumors.²⁵ They are noted in approximately 2% of individuals in autopsy series.²⁶ The annual incidence is approximately 2 to 7 out of 100,000 for females and 1 to 5 out of 100,000 for males.²⁷ There is a peak incidence in the fourth through sixth decade of life and an approximate 2:1 female:male predominance. They arise from the arachnoid cap cells imbedded in the arachnoid villi, and they can occur in any location but arise most commonly at the skull vault and skull base. Less common sites include the optic nerve sheath, choroid plexus, and spinal cord. They are most often classified according to their dural site of origin, the involvement of adjacent tissues (e.g., venous sinuses, bone, brain, and nerves), and the World Health Organization (WHO) histological grading schema. Ninety percent of meningiomas will be classified as WHO grade I. Less common histology subtypes include atypical (WHO grade II) meningiomas (5–7%) and anaplastic (WHO grade III) variants (1–3%). The clinical presentation, regardless of the grade, will generally be dependent on the location of the mass. Since most meningiomas are slow growing, the majority of patients will have a relatively insidious onset of symptoms including headache, pain, subtle personality change (often confused with dementia or depression), and less commonly seizure.²⁵ Rarely, meningiomas may present with hemorrhage that may be intratumoral,^{28,29} intraparenchymal^{28,29}, intraventricular^{16,30}, or subarachnoid.³¹ Cheng and Lin²⁸ noted a 55% mortality in cases of spontaneous meningioma hemorrhage, and stressed that early cross-sectional imaging and surgery improved survival. Spontaneous meningioma hemorrhage has not been shown to have an association with other known factors for cerebral hemorrhage including hypertension, blood dyscrasia, atherosclerosis, and

anticoagulant use. Three reviews on the subject showed no association of hemorrhage with the age or sex of the patient.^{32–34} One study found a higher proportion of convexity and intraventricular meningiomas, as well as angioloblastic subtypes, among hemorrhagic meningioma in relation to their relative prevalence in meningiomas in general.³³

Meningioma—Treatment Options

Treatment for meningiomas is based primarily on symptoms. Meningiomas detected incidentally on imaging generally show slow or no growth.^{35–37} As the majority of these will remain asymptomatic, it is generally held that these can be watched clinically and by imaging. Factors associated with higher rates of growth include lack of calcification, large size, and younger age patients.³⁵ For symptomatic meningioma, the treatment of choice is open surgical resection, which may be curative if complete resection can be achieved. Completeness of resection has been traditionally graded using the Simpson grading system (see ►Table 1). Simpson grade I patients traditionally have a 9% 10-year recurrence rate as compared with Simpson grade III patients who have a 29% 10-year recurrence rate.³⁸ Models to predict the likelihood of recurrence now include the postoperative 72-hour brain CT/MR findings, and the histopathological findings or WHO grade from the resection specimen. Despite advances in neurosurgery over the past several decades, complete resection will not be achieved in many cases due to the meningioma location or local tissue invasion.

Radiation (conventional radiotherapy, radiosurgery, etc.) is generally used to treat patients with incomplete resection, postresection cases with atypical or malignant histology, and those not amenable to surgery. Goldsmith et al reported an 89% 5-year progression-free survival rate in 140 patients with partially resected meningioma treated with adjunctive conventional radiotherapy.³⁹ Radiosurgery is increasingly being used for meningiomas to limit dose to normal tissue and allow higher doses of radiation to be delivered more precisely. Radiosurgery is often used in the same cases that conventional radiation is used, and has also shown promise as a primary treatment.⁴⁰ Simpson grade I tumors are controlled with radiation therapy in 91% of patients at 10 years.

Table 1 Simpson classification meningioma

Simpson grade	Completeness of resection	10-year recurrence (%)
Grade I	Macroscopic complete removal including resection of underlying bone, associated dura, and venous sinus if involved	9
Grade II	Macroscopic complete removal + coagulation of dural attachment	19
Grade III	Macroscopic complete removal of intradural tumor w/o resection or coagulation of dura/ dural attachments	29
Grade IV	Partial removal or subtotal resection leaving macroscopic intradural tumor	40
Grade V	Simple decompression, with or without biopsy	100

Overall, chemotherapy has shown little effect against meningioma. Hydroxyurea, one of the better tolerated agents, has demonstrated modest activity against meningioma. It has been used for unresectable tumors and those involving the skull base and/or dural sinus. For malignant meningioma, following surgery or radiation, adjuvant CAV (cyclophosphamide, Adriamycin (Adriamycin-Pharmacia and Upjohn Company, Mississauga, ON, CA), and vincristine) therapy has been used to slow down progression, with median time to tumor progression of 4.6 years and median survival of 5.3 years.⁴¹ Hormonal therapies have been attempted with little convincing evidence of benefit to date.³⁸

Preoperative Embolization of Meningioma—Overview

Embolization of meningiomas was first described by Manelfe et al in 1973.² Since that time there have been numerous reports using embolization as a preoperative adjunct,^{1,3,6,9,11,12,15,17,18,20,22,24,42–46} several reports of primary treatment in nonoperative cases,^{3,13–15,19} and rare descriptions of urgent treatment to control acute hemorrhage.¹⁶ Over the past 30 years, there have been numerous improvements in digital subtraction angiography (DSA) and microcatheter technology, which, when used appropriately, make distal vessel treatment safer and easier. Despite these advances, the IR must recognize the risk associated with the procedure.^{4,8,11,15,21,22,24,32,47,48} Safe embolization of meningioma is no different than safe

embolization of a tumor elsewhere in the body: it relies upon a thorough knowledge of pertinent anatomy (including potential collaterals and cranial nerve [CN] supply), careful analysis of tumoral blood supply, skillful catheterization of feeding vessels, accurate delivery of an appropriate embolic agent, and an understanding of patients most likely to benefit from embolization. Each of these areas deserves careful consideration and discussion to maximize the efficacy and safety of embolization.

Anatomical Considerations

Meningioma blood supply may arise from the external carotid artery (ECA), internal carotid artery (ICA), vertebral artery (VA), or any combination of these vessels. Classically, the central region of a meningioma is supplied by dural feeders at the site of dural attachment, while the capsule is fed by pial or cortical feeders. ECA branches often supplying meningioma include the middle meningeal artery (MMA), accessory meningeal artery (AccMA), superficial temporal artery, ascending pharyngeal artery (APA), and perforating transosseous occipital artery (OccA) branches. Dural ICA branches commonly supplying meningiomas include those arising from the meningohypophyseal trunk (MHT), the inferolateral trunk (ILT), and the ophthalmic artery (OPA). The VA often supplies the dura via its posterior meningeal artery (PMA) branch. One must appreciate both common and uncommon ECA–ICA communications as well as the potential for CN supply when working in this area (►Table 2). Because the

Table 2 Important vascular communications

External carotid artery (ECA)		Internal carotid artery (ICA)		Other supply
Parent vessel	Branch(es)	Branch(es)	Parent vessel	
STA	Frontal	Supraorbital artery	OpA	
APA (anterior)	Inferior tympanic	Caroticotympanic	Petrous ICA	MMA via PS
APA (anterior)	Superior pharyngeal	ILT, AFL	Cavernous ICA	
APA (anterior)	Pharyngeal			IMAX via AccMA, DPA
APA (posterior)	Jugular	Clival branches	MHT	Cr N IX, X, XI
APA (posterior)	HG	Clival branches	MHT	Cr N XII
APA (posterior)	HG or MS branches			VA, OccA
Posterior auricular	Stylomastoid			Cr N VII, VII
MMA	PS	Lateral clival	MHT	Cr N VII, VII
MMA	Frontal	Lacrimal, AFA, ReMA	OpA	
MMA	various	Dural	ILT	
IMAX	AccMA	Posteromedial branch	ILT	Trigeminal ganglion
IMAX	Anterior tympanic	Caroticotympanic	Petrous ICA	
IMAX	Vidian	Vidian	Petrous ICA	
IMAX	AFR	Small comm. artery	MHT, ILT	
IMAX	AFO	Small comm. artery	ILT	

Abbreviations: AccMA, accessory meningeal artery; AFA, anterior falcine artery; AFL, artery of foramen lacerum; AFO, artery of foramen ovale; AFR, artery of foramen rotundum; APA, ascending pharyngeal artery; DPA, descending palatine artery; HG, hypoglossal branch; ILT, inferolateral trunk; IMAX, internal maxillary artery; MHT, meningohypophyseal trunk; MS, musculospinal branch; OpA, ophthalmic artery; PS, petro-squama; ReMA, recurrent meningeal artery; STA, superficial temporal artery; VA, vertebral artery.

dominant supply to a meningioma is primarily related to its location, it is useful to consider the important anatomy of common locations when considering embolization.

Frontal Region/Anterior Cranial Fossa

The MMA (discussed below) and anterior falcine artery (AFA) often supply anterior parasagittal and high convexity meningiomas. The AFA supplies the dura of the anterior cranial fossa; it arises from the anterior ethmoidal artery (EthA), a branch of the OPA. The recurrent meningeal artery (ReMA) branch of the OPA supplies the anterior basal meninges and anterior middle cranial fossa (MCF). The AFA and EthA frequently supply frontal-polar and falcine tumors. Parasagittal meningiomas often have bilateral MMA supply. Meningiomas of the olfactory groove will commonly be supplied by the anterior and posterior ethmoidal arteries.⁴⁹ Although successful embolization of OPA feeders has been described,^{10,24,50} it is generally regarded as too risky unless the eye is already blind.

Middle Cranial Fossa

Branches of the internal maxillary artery (IMAX), including the MMA, AccMA, artery of foramen rotundum (AFR), and vidian artery (VidA), commonly supply MCF meningiomas along with branches from the APA and OccA.⁴⁹ The MMA, because it is so often the target of embolization, deserves special attention. The MMA is generally the largest and most proximal branch of the IMAX. It generally travels superiorly and makes a sharp medial turn at the foramen spinosum where it enters the skull. It has frontal, parietal, and petrosal branches. It provides dural supply to a large portion of the meninges and anastomoses with other meningeal arteries potentially including the AFA and MHT. The petrous branch provides supply to the seventh CN. The MMA may collateralize to the OPA through the ReMA, lacrimal artery, or via its anastomoses with the AFA. Rarely, the OPA may arise directly from the MMA. The AccMA may arise from the IMAX or MMA. It is primarily external but has a small branch that pierces the calvarium to supply the dura of the temporal fossa and to the gasserian ganglion. Both the MMA and AccMA may collateralize to the ICA via communications with the ILT. Branches of the MMA and the ReMA will frequently supply meningiomas of the sphenoid wing and paraclinoid region.

ICA blood supply in this region is also worthy of discussion. The petrous segment of the ICA gives rise to the carotico-tympanic artery (CCTA), which anastomoses with the anterior tympanic artery from the IMAX and inferior tympanic artery (ITA) from the APA. The petrous ICA may give rise to the VidA (more often arises from the IMAX), which can communicate with IMAX. The cavernous segment of the ICA gives rise to the MHT and ILT; the MHT originates as a single trunk or a collection of vessels from the proximal cavernous ICA. It supplies the posterior pituitary, portions of the clivus, CN III–VI, portions of the tentorium cerebelli, and adjacent dura. Specifically, the MHT contributes to the cavernous segment of CN IV and to CN VI in the region of Dorello's canal. Named branches of the MHT include the marginal tentorial artery (aka, artery of Bernasconi and Cassinari), the dorsal menin-

geal artery, and the inferior hypophyseal artery. Extensive collateral pathways exist with the ILT, MMA, and hypoglossal branch of the APA. The ILT arises from the cavernous ICA more distally and supplies the gasserian ganglion, CN III–VI, and the wall of cavernous sinus and inferior petrosal sinus. The ILT may collateralize to or give some supply to the MHT distribution; it gives rise to or communicates with the AFR and artery of foramen ovale. Anterior branches anastomose with the OPA.

Posterior Cranial Fossa

The dural region of the posterior fossa is supplied by various regional arterial branches, the largest of which is the PMA. The PMA typically arises from the suboccipital portion of the VA but may also take origin from the OccA or APA. The PMA enters the cranium through the foramen magnum and ascends along the lateral aspect of the occipital bone. Posterior fossa meningiomas are primarily supplied by the PMA as well as branches of other arteries including the APA, MMA, AccMA, and OccA. The PMA and, when anteriorly located, anterior meningeal artery supply foramen magnum meningiomas. The OccA supplies the meninges of the posterior fossa via the artery of the falx cerebelli and mastoid branch. It may collateralize to the VA via these branches. Petroclival meningiomas are supplied branches of the MMA, OccA, posterior auricular artery, anterior inferior cerebellar artery, and APA. Tentorial branches of the MHT, ILT, MMA, and AccMA generally supply tentorial meningiomas.^{49,51}

The APA is worthy of discussion due to its frequent supply to meningiomas, potential to communicate with the VA and ICA, and significant CN supply. The APA has an anterior and a posterior branch. The anterior (pharyngeal) branch supplies primarily the pharynx and gives rise to the ITA. This pharyngeal branch may communicate with the ICA via the superior pharyngeal branch or via the ITA to the CCTA. The posterior division is referred to as the neuromeningeal branch, which subdivides into two branches. The jugular branch traverses the jugular foramen and provides blood supply to CN IX–XI. The hypoglossal branch traverses the hypoglossal canal and supplies the hypoglossal nerve and meninges in the area of the odontoid. Both divisions of the neuromeningeal branch anastomose with clival branches of the MHT. Anastomoses with the VA are present in the 2nd and 3rd cervical levels.

Additional contributions to the CNs arise from other branches of Occ, APA, and posterior auricular arteries. The proximal segments of CN VII and VIII are supplied by the labyrinthine artery, while more distally they are supplied by the petrous branch of the MMA and the stylomastoid branch of the posterior auricular artery. The cisternal portions of the lower CNs are supplied from the ipsilateral VA and their foraminal portions are supplied mainly by the neuromeningeal trunk of the APA.

Basic Technique

Meningioma embolization begins with standard femoral access in the majority of cases. If the femoral artery is not available, radial, brachial, or even carotid access can be used.

Figure 1 Large parieto-occipital meningioma. (A) Lateral scout image shows marked hyperostosis (arrows) occasionally seen with large meningioma. (B) lateral arch injection study shows markedly enlarged bilateral occipital arteries (arrows). (C) Right ECA injection shows extensive hypervascularity from multiple MMA (1) branches (long arrows) as well as STA (2) branches (wide arrows). This is different from the (D) left ECA injection that shows primarily MMA supply (long arrows). Effective preoperative embolization required treatment of the right MMA and STA feeders, left MMA feeders, and bilateral occipital embolization. ECA, external carotid artery; MMA, middle meningeal artery; STA, superficial temporal artery.

Premedication with steroids are recommended by many authors.^{11,15} Selective mapping arteriography with 5 Fr diagnostic catheters is generally performed initially. Bilateral arteriography will be required for lesions abutting the midline (see ►**Fig. 1**) and for many skull base lesions. Many physicians will heparinize patients either at the beginning of the case or before more selective catheterization.^{24,50}

Once initial mapping of local anatomy and general supply to the meningioma are assessed, most physicians will place a 5- or 6-Fr soft-tip guiding catheter into the common carotid arteriography, ECA, or ICA (depending on the vessels being chosen for further evaluation) to allow control catheter runs while the microcatheter is in place. A microcatheter is introduced to allow selective catheterization of the vessels to be studied. Vasodilators may be used to assist in preventing and/or relieving spasm, which often occurs when catheterizing the ECA and its branches. Many meningiomas will demonstrate a characteristic angiographic pattern of enhancement called the “mother-in-law sign,” where the arterial blush is seen very early in the arterial phase, increases and densely enhances throughout, and persists well into the venous phase (“arrives early and stays late”) (see ►**Fig. 2**). Some meningiomas will be relatively hypovascular arteriographically, in

which case embolization is unlikely to provide any benefit (see ►**Fig. 3**). It is also important to note the venous drainage as well as the patency of the sinus(s) adjacent to the meningioma. The patency of dural sinuses (see ►**Fig. 4**) and pathways of secondary collateral venous flow should be assessed, as these factors are critical to surgical decision making. Consideration of both visualized and potential ECA-ICA anastomoses and important CN supply should be undertaken prior to embolization. A summary of important vascular communication is provided in (►**Table 2**). Provocative testing with a small intra-arterial dose (10–20 mg) of 2% cardiac lidocaine, followed by neurologic testing, should be performed if there is concern about CN supply.^{7,52}

Embolization is performed slowly with the primary intent of preserving antegrade flow for as long as possible during the embolization. Embolization is performed until there is loss of tumoral blush as well as decreased blood flow toward the tumor (see ►**Figs. 2, 5, and 6**). Special caution is advised during the embolization, as it is a dynamic process where target vessels are being occluded, while nontarget vessels and collateral pathways may be opening in response to the changes resulting from the embolization. Rarely, as described by Carli et al,¹⁵ one may see contrast extravasation during

Figure 2 (A) Lateral contrast-enhanced MR shows homogeneously enhancing middle cranial fossa meningioma. (B) Lateral CCA injection shows no significant CCA supply. (C) Early ECA injection shows classic early arterial enhancement typical of meningioma, which intensifies in mid-arterial phase (D), and persists late into venous phase (arrows) (E). (F) Distal microcatheter selective injection (arrowhead) shows filling of meningioma and MMA branches. (G) Postembolization angiogram demonstrates near-complete devascularization following injection of 150–250 μ m PVA. CCA, common carotid arteriography; ECA, external carotid artery.

embolization. This is described as a slow collection of contrast in or around the tumor. It may occur toward the end of the embolization, during or after stagnation of the contrast agent in the feeding artery, and without overt arterial perforation. Urgent surgery may be lifesaving in such cases.¹⁵

Embolization Agent of Choice

Many different agents have been used during embolization of meningiomas. The most common embolic agents include Gelfoam (GF) (Gelfoam-Pharmacia and Upjohn Company, Kalamazoo, MI)^{1,5,9,42,46} and polyvinyl alcohol (PVA).^{3,5,7,9,11,15} A few more recent series describe the use of calibrated microsphere embolics including calibrated tris-acryl microspheres (Embosphere [ES], Merit Medical, South Jordan, Utah^{3,53}; and Embozene [EZ], Celo-Nova Bioscience,

Ulm, Germany).⁴⁸ There are also descriptions of other agents including lyophilized dura mater (lypo-dura),^{20,46} as well as liquid embolics including alcohol,⁵⁴ N-butyl cyanoacrylate (NBCA),⁴⁴ and Onyx (Ev3/Covidien, Plymouth, MN).⁵⁰ Following tumor embolization, some authors (usually when the MMA is involved) recommend placing an occlusive microcoil into the feeding artery proximal to the tumor bed, reporting that it permits easier surgical transection of the artery.^{6,55} GF and PVA are the most widely used embolics and are favored by most physicians due to their long-track record of safe use.

The size of the embolic agent will depend on the local anatomy (vessel size, tortuosity, risk of nontarget embolization), microcatheter used, and the desired goal of the procedure. The goal in most cases will be to deliver embolic agents to the tumor without penetrating deeply enough to occlude the outflow vessels. Occlusion of outflow has been postulated

Figure 3 Symptomatic 5.2-cm cribriform plate meningioma in a 78-year-old patient. (A) AP and (B) lateral left ICA injections show mild hypervascularity (arrows) of the meningioma with some supply from anterior falcine artery and recurrent meningeal artery. Meningioma in this location will often have ethmoidal artery supply. (C) AP and (D) lateral left ECA injection shows virtually no supply to the meningioma. The right-sided injection showed similar appearance. The lack of hypervascularity and risk of catheterization of small ICA branch feeders resulted in decision not to perform embolization in this case. The patient underwent surgery without preoperative embolization. AP, anteroposterior; ECA, external carotid artery; ICA, internal carotid artery.

as one potential cause of hemorrhage following embolization.²² Conversely, occluding feeding arteries too proximally is avoided to prevent recruitment of contralateral or other alternative collateral supply.⁴⁴ Reports describe the use of GF powder^{5,7,9,42} or pledgets ranging in size from 40–60 μm to 1 mm, respectively. PVA of varying sizes has been utilized, primarily including 45 to 150 μm ,^{3,7,11,15} 150 to 250 μm ,^{3,11,15} and less commonly 250 to 350 μm ²⁴ or 350 to 500 μm ²⁴ sized particles. The smaller the embolic used, the more deeply it will penetrate the meningioma and the higher the degree of necrosis should be expected.^{5,11,42} Some authors⁹ recommend that particles no smaller than 100 μm (or even >250 μm) be used in areas where there are either potential dangerous collaterals to the ICA or supply to the nerva vasorum of CNs.

Wakhloo et al conducted an elegant study comparing the use of 150–300 μm (group 1) to 50–150 μm PVA (group 2).¹¹ The study demonstrated significant differences between the two groups (large vs. small particles) in the following ways: (1) MRI/CT hypoperfusion/necrosis (14 vs. 100%, respectively); (2) MR volume change (0 vs. 35%, respectively); (3) pathological necrosis (14 vs. 100%, respectively); (4) embolic material noted in precapillary bed (0 vs. 75%, respectively);

and (5) blood loss at surgery (846 vs. 305 mL, respectively). Most surgeons reported “some degree of decrease in vascularity” in the majority of large particle group patients versus “visible devascularization” in most cases and macroscopic necrosis in 40% of small particle cases. A single symptomatic patient from each group had improvement in symptoms and did not undergo surgery. The 10 to 25% MRI volume increase in three small particle patients led to mild transient symptoms in one patient that resolved after surgery. The authors suggested that in all these cases, inadequate steroid had been given. A single patient from the large particle group had acute postembolization hemorrhage requiring surgery but recovered without deficit. In a separate study, Carli et al reported on 198 patients treated with either 45 to 150 μm or 150 to 250 μm PVA.¹⁵ Their study describes a 5.6% rate of hemorrhage, which generally required emergent surgery, and a 3.5% rate of death or major disability related to embolization. The only positive risk factor for complications was the use of smaller particle (odds ratio, 10.2; confidence interval, 1.3–80.7; $p = 0.027$).

Bendszus et al³ compared the use of 100 to 300 μm calibrated tris-acryl spheres (ES) (group 1) to both 45 to 150 μm (group 2) and 150 to 250 μm (group 3) PVA in a total of

Figure 4 (A) Lateral and (B) frontal postcontrast MR images depict large right frontal meningioma with significant mass effect on adjacent structures. (C) AP and (D) lateral right CCA injections show mass effect and displacement (arrows) on anterior cerebral artery. (E) Lateral left CCA injection shows supply from enlarged anterior falcine artery (arrow). (F) Late phase image from left CCA injection shows non-filling of anterior half of superior sagittal sinus (arrows). Patency of sinuses is important to note for surgical planning. AP, anteroposterior; CCA, common carotid artery.

60 patients. They reported no significant differences in the angiographic degree of devascularization, but found significant reductions in intraoperative blood loss between group 1 and both PVA groups (621 vs. 881 vs. 917 mL; $p < 0.05$). A nonsignificant trend toward decreased transfusion was also noted for group 1. Group 1 showed more distal tumor penetration than either PVA group, a finding that was highly significant ($p < 0.005$). This finding is in keeping with animal data that show, for a given vessel of a given size, ES penetrate significantly deeper than PVA of

comparable size.⁴³ The use of smaller PVA in group 2 patients also showed more distal penetration compared with group 3, in keeping with the findings of Wakhloo.¹¹ Complications are not disclosed in this study. In another study, Rodiek et al⁵³ reported a series of 17 patients undergoing preoperative embolization using 40 to 500 μ m ES and showed necrosis in 77%, with the most distal tumoral penetration by the smallest particles.

A recent study describes the use of a newly available embolic agent called EZ. These microspheres consist of a

Figure 5 (A) AP projection MMA branch injection in patient with large parasagittal meningioma. (B) Lateral MMA injection. (C) Postembolization appearance following injection of 150 to 250 µm PVA. AP, anteroposterior; MMA, middle meningeal artery; PVA, polyvinyl alcohol.

hydrogel core of polymethylmethacrylate and a flexible shell of polyphosphazene. Calibrated spheres of 400 µm were administered to 55 patients. Comparison was made to a cohort of patients who had been treated with either 45 to 150 µm PVA ($n = 108$) or 150 to 300 µm PVA ($n = 93$). No ischemic or hemorrhagic complications were noted in the EZ group, whereas there were 8 out of 108 (8.3%) such complications in the small PVA group ($p = 0.06$) and 1 out of 93 (1.1%) in the large PVA group ($p = 0.8$). Blood loss, transfusion requirement, and ease of operation were not reported; however, “neither we nor our neurosurgeons noticed any difference between PVA and microspheres.”

Limited reports of liquid embolics are available in the literature, and therefore few statements can be made regarding their use. Kominami et al⁴⁴ reported using NBCA in 31 patients with meningioma. Forty-six arterial pedicles were catheterized and it was deemed safe to use NBCA in 38 (83%). PVA or GF was used in cases where safe catheterization was not possible. Eighty-five percent of vessels treated were ECA branches as well as a small number of ophthalmic, ICA, and VA branches. Total devascularization was achieved in 3 patients, near-total in 15 patients, and greater than 50% in 11 patients. Complications included proximal occlusion of the MMA in one patient with resultant feeding of tumor by inaccessible collaterals; VA dissection and brainstem infarct occurred in another one patient. The use of Onyx has been described in a small series of five patients treated for meningiomas with OpA supply.⁵⁰ The microcatheter was placed beyond the central retinal artery as close to the tumor as possible. Angiographic devascularization was described as “complete” in two patients and “extensive” in three patients. One case of oculomotor nerve paralysis was noted for a complication rate of 20%. Surgery was successful in all patients with “minimal blood loss during the operation” and no transfusions.

Postprocedure Appearance

Following embolization most authors report a lack of angiographic enhancement of the meningeal supply to the tumor, as well as slowing or cessation of flow within the feeding

artery (see ►Figs. 2, 5, and 6). Extravasation of contrast, if seen, has been associated with hemorrhage on follow-up CT and is an ominous sign.^{5,9,11,12,15,20} In cases where there are other supplying arteries (ECA or ICA branches) that cannot be safely embolized, one will generally see residual enhancement of at least a portion of the tumor (see ►Fig. 3).

Cross-sectional imaging studies following embolization have a variable appearance. Manelf et al⁹ reported that in 50% of CTs done within 72 hours of embolization, new areas of low density thought to represent necrosis could be seen within the meningiomas and, paradoxically, pathological correlation was imperfect in two-thirds of cases, and the low density on CT did not correlate with necrosis on pathological examination at the time of surgery. Furthermore, large areas of infarction were noted despite normal CT, although this latter finding may be related to delay between CT and surgery or changes induced during the surgery. In a separate study, Teasdale et al⁴⁶ found CT hypodensities in 8 out of 17 (47.1%) scanned patients who later went on to surgery embolized with either 0.5×0.5 mm GF or similar size lipo-dura. Seven of eight (87.5%) patients with CT hypodensity were noted to have exclusive ECA supply. At pathological exam following surgery on 26 patients, the authors reported necrosis in 14 patients (53.8%). Carli et al reported major edema and midline shift in 27% of 201 embolized meningiomas on follow-up CT performed within 24 hours.¹⁵ Hemorrhage was noted in 5.6% (11/201). Finally, Wakhloo et al reported edema and increase in tumor size in 15% of patients treated with 50 to 150 µm PVA as well as hypoperfusion or necrosis in 100%. These changes were not seen in patients treated with 150 to 250 µm PVA particles, suggesting that the postembolization appearance is related to the size of the embolic¹¹ (►Table 3).

As no well-controlled large studies exist, it is not possible to say with certainty which is the correct agent to use in any setting. Smaller particles penetrate more deeply into the meningioma and most often cause more significant necrosis. The link between necrosis, blood loss, transfusion requirement, and ease of surgery is less clear. Spherical embolics (ES, EZ) appear to penetrate more deeply and act like smaller

Figure 6 Multiple images from AP and lateral ICA and ECA arteriogram from patient with right fronto-parietal meningioma. Selective AP (A) and lateral (B) right ICA injection images show no distinct ICA supply. Arrows point to area of hypovascularity likely related to mass in this area. Selective AP (C) and lateral (D) ECA injection images show moderate meningioma hypervascularity (large arrows) supplied by the anterior branches of right middle meningeal artery (small arrows; A; P = posterior branches). (E) Selective right MMA (arrow) injection following embolization with 150 to 250 μ m PVA particles. AP, anteroposterior; ECA, external carotid artery; ICA, internal carotid artery.

size PVA. It is not clear if their use, like the use of 45 to 150 μ m PVA,¹⁵ is more prone to hemorrhage following embolization. The role of liquid embolics remains undetermined. Given these limitations, the choice of embolic should be based on the vessel selected, the angiographic findings, and experience of the physician.

Timing of Surgery

The timing of the surgery following embolization varies in the literature from 1 day¹⁵ to more than a week.^{3,4,11} In

most cases, the surgery is performed during the same hospital stay and within 72 hours of the embolization.⁷ Djindjian recommended an interval of 3 days,¹ but Richter and Schachenmayr reported no difference at surgery in patients treated between 1 day and 2.5 months after embolization.²⁰ Bendszus et al performed a regression analysis to investigate the effect of time between embolization and surgery and found no association with the extent of blood loss.³ Kai studied the effect of embolization on 42 patients and reported the “greatest degree of tumor

Table 3 Summary of studies examining different embolics

Year	Author	N	Embolic	Complications				Comments
				Any	Bleed	Perm Neuro	Death	
1993	Wakhloo et al	20	50–150 PVA	5%	0%	0%	0%	Necrosis 100%, edema 50%, visible devascularization all cases, 5% symptomatic improvement w/o surgery
		14	150–300 PVA	7%	7%	0%	0%	Necrosis 14%, edema 0%, some decrease vascularity most cases, 7% symptomatic improvement w/o surgery
2000	Bendszus et al	15	45–150 PVA	NR	NR	NR	NR	Significantly more distal penetration c/w PVA 150–300
		15	150–300 PVA	NR	NR	NR	NR	Least distal penetration, no significant difference in inflammatory reaction or angiographic extent of devascularization
		30	150–300 ES	NR	NR	NR	NR	Significantly lower intraoperative blood loss, nonsignificant trend to lower transfusion, Significantly more distal penetration into tumor, NS trend toward greater necrosis
2010	Carli et al	108	45–150 PVA	NR	8.3%	1%	3.5%	Overall complication rates given for permanent neurologic deficit and death, Significantly higher complications with use of 45–150 PVA
		93	150–300 PVA	NR	1.1%			
2013	Sluzewski et al	55	400 EZ	0%	0%	0%	0%	Significantly fewer complications c/w 45–150 PVA group ¹⁵

Abbreviations: c/w, compared with; ES, Embosphere; EZ, Embosphere; NR, not reported; NS, not significant; PVA, polyvinyl alcohol.

softening” between days 7 and 9.⁵⁶ Chun et al⁶ compared immediate surgery (< 24 hours) with delayed surgery (> 24 hours), and found significant reduction in blood loss (475 vs. 337 mL; $p = -0.01$); a positive correlation with tumor size and blood loss was noted in the immediate group that was not seen in the delayed group ($p = 0.03$). Eight patients in the immediate group had blood loss greater than 1,000 cc, compared with the greatest blood loss of 700 cc in the delayed group. No difference in the extent of necrosis at histological examination was noted between the groups, a finding that was also reported by Teasdale et al.⁴⁶ The authors postulate that necrosis may not be the sole determinant in the ease of resection and that other factor such as tumor consistency and texture may play a role. Intervals greater than 1 week may allow potential for recanalization or collateralization^{14,57}; therefore, most centers perform surgery within 7 days.

Patients Most Likely to Benefit

It has been suggested that not all meningiomas will need preoperative embolization. Certain locations and “challenging” meningiomas represent higher surgical risk and are more often evaluated for possible embolization.³¹ Challenging meningiomas include those located at the sphenoid wing,⁴ MCF,^{9,31} para-cavernous,⁹ as well as those with tumorous involvement of the sinus,³¹ large convexity,^{4,31} or invading bone or soft tissue.^{4,31} MCF meningiomas present a special challenge as the arterial supply is deep and not reached until the late phases of tumor resection. Patients who are at high anesthetic risk may also benefit from embolization. In some cases, where the risk of surgery is prohibitive, embolization may be offered as a primary treatment.^{13–15,19}

The source and degree of blood flow to the tumor is especially important in determining the value of embolization. Manelfe et al⁹ and Richter and Schachenmayr²⁰

suggested that meningiomas be divided based on their predominant blood supply: (1) those with sole ECA supply, (2) those with mixed ECA/ICA supply but dominant ECA feeders, (3) mixed ICA/ECA but dominant ICA supply, and (4) those with exclusive ICA supply. Patients most likely to benefit from embolization fall into groups 1 or 2. Although conventional DSA remains the gold standard for assessment of blood supply, both CT⁵⁸ and MR^{59–61} show promise as adjuncts in determining blood supply and perfusion characteristics of meningioma. Although available until recently at only a few centers, Hirai et al reported that angio-CT was superior to DSA in detailing meningioma blood supply.⁶²

Most authors agree that unless embolization will be the primary therapy and surgery is not planned, it makes little sense to embolize meningiomas with limited ECA supply and dominant ICA supply.^{15,20} Aside from a few highly specialized centers with dedicated neurointerventionalists,^{24,44,50,63} routine preoperative embolization of ICA branches is not recommended due to the inherently increased risk of ICA branch embolization. If the blood supply is ICA dominant, embolization of a small ECA contribution will be of no utility. The maximal benefit occurs in cases where there is pure or predominant ECA supply.⁹ Teasdale et al⁴⁶ reported that in the 13 out of 26 patients in whom benefit was shown, 70% had pure ECA supply; of those with persistent bleeding at surgery or lack of perceived benefit, only 1 had a pure ECA supply. Furthermore, several authors^{20,46} report that embolization of ECA branches in cases with significant ICA supply may actually lead to increased ICA supply.^{1,46} There remains debate on this final point with other authors reporting a beneficial effect of ECA branch embolization in such cases with no increase in blood supply from ICA branches.^{9,11} ► **Table 4** summarizes characteristics of meningioma most likely to benefit from preoperative embolization.

Comparisons of Surgery with and without Embolization

Macpherson reported a personal experience with 28 embolized and 24 nonembolized meningiomas. He suggested a beneficial reduction in blood loss and surgical complications with improved outcomes and subjective reports of easier surgery according to the neurosurgeon.¹² Overall complications were 21% in the preoperative embolization group versus 58% in the operative-only group. Dean et al⁷ described a matched pair study of 18 patients where they found signifi-

cant reductions in intraoperative blood loss ($p = 0.048$) and number of transfusions ($p = 0.041$) in the group undergoing embolization. Although trends in reductions in length of surgical procedure, length of hospital stay, and overall hospital costs were noted, these variables did not reach statistical significance. No permanent neurological complications related to embolization were reported.⁷ Bendszus et al³ reported on a prospective study of 60 patients treated at two centers, one implementing routine preoperative embolization and the other not. They described no statistically significant difference in mean blood loss, need for transfusion, or rates of surgical morbidity. There was also no objective difference in the neurosurgeons impression of hemostasis, tumor consistency, and intratumoral consistency. There was a significant difference in intraoperative blood loss in the embolization patients with > 90% devascularization as assessed by MRI; however, no significant reduction in units of blood transfused was seen. ► **Table 5** summarizes these studies.

Embolization as a Stand-Alone Therapy

In some cases, where the risk of surgery is prohibitive or there is symptom relief following embolization, embolization has served as a primary treatment.^{13–15,19} Koike et al described the use of 250 to 500 μ m PVA in an initially symptomatic 73-year-old patient. When the dominant symptoms resolved, the patient refused surgery. CT at 10 months showed 60% volume reduction. Clinical follow-up up to 4 years documented no change in neurological status with CT showing slight interval regrowth. Das and Singh¹⁶ described primary PVA (size not mentioned) embolization in a highly compromised 62-year-old patient presenting with acute intraventricular and intraparenchymal hemorrhage and deemed not fit for surgery. She made a good but incomplete neurological recovery, and 6 months follow-up CT showed decreased tumor size. Bendszus et al described stand-alone meningioma embolization (100–300 μ m ES) in a series of seven patients. They reported tumor shrinkage in 86%, stable tumor size in 1 patient, and symptom control in 100% at mean follow-up of 20 months.¹³ Carli et al¹⁵ included 28 patients treated primarily with embolization in a larger series of patients treated with preoperative embolization; however, discrete follow-up on this subset of patients was not reported. Clinical and imaging follow-up of such patients is indicated to evaluate the potential for continued tumor growth, which may occur even after several years of shrinkage.¹⁹

Table 4 Meningioma most likely to benefit from embolization

Tumor size	Tumors > 5 cm in general are more difficult surgically, thus more benefit to embolization
Location	Sphenoid wing, middle cranial fossa, paracavernous, involving dural sinus or bone
Blood supply	Exclusive or dominant ECA supply
Vascularity	Hypervascularity greater than adjacent tissue
Supplying vessel	Sufficient size to allow microcatheter placement, lack of supply to CN or ICA

Abbreviations: CN, cranial nerve; ECA, external carotid artery; ICA, internal carotid artery.

Table 5 Summary of studies comparing meningioma surgery with and without preoperative embolization

Year	Author	N	Group	Complications		Mortality		Blood loss	Transfusion	Comments
				Embo	Surgery	Embo	Surgery			
1991	Macpherson	28	Embo	18%	21%	0	7%	Reduced	Reduced	Good outcome: 78% embo vs. 58% nonembo
		24	Nonembo	–	54%	–	8%	–	–	
1994	Dean et al	18	Embo	22%	33%	0	0	Reduced	Reduced	Blood loss: $p = 0.048$ Transfusion: $p = 0.041$
		18	Nonembo	–	67%	–	5.5%	–	–	
2000	Bendszus et al	30	Embo	3%	17%	0	0	NS	NS	Blood loss reduced only if > 90% devascularization achieved on post-embo MR
		30	Nonembo	–	20%	–	0	–	–	
2009	Quiñones	45	Embo	2.2%	NR	0	0	NR	NR	Gross total resection of > 5 cm tumor 10x more likely if embolization could be performed ($p < 0.006$)
		23	Nonembo	–	–	–	–	–	–	

Abbreviations: NS, not significant; NR, not reported.

Complications of Embolization

The overall risk of meningioma embolization ranges from 0% to 8.3%.^{4,6,15,44,48} Risks include those of arteriography (groin hematoma, contrast reaction, vessels dissection, etc.), which are generally mild, as well as more serious sequelae associated with neuroembolization. Such complications include thromboembolism related to catheterization or misembolization, CN palsy due to vasa nervorum injury (more often related to the use of smaller particles or liquid embolics), intratumoral or peritumoral hemorrhage that may be related to necrosis or rupture of small vessels, skin or scalp necrosis related to embolization of skin branches, and postembolization swelling and edema leading to mass effect. Death and/or permanent disability are very uncommon.

The risk of hemorrhage following meningioma embolization deserves special attention. Due to the potential need for urgent surgery in such rare cases, all practitioners should be aware of this risk and be ready to act quickly should it occur. Although the literature describes a total of 20 cases to date,^{11,15,21–23,32,50,64,65} it is difficult to know the true incidence. The phenomenon of intra- and peritumoral hemorrhage, with or without subarachnoid hemorrhage, was initially described in scattered case reports between 1986 and 1993.^{11,32,47,64,66} A review by Kallmes et al²² described seven cases in the world literature in 1997. Most cases involved a relatively complete devascularization via the MMA, and hemorrhage tended to occur immediately after embolization. The authors found the only commonality between cases was a large tumor (6 cm or greater) and dense hypervascularity on arteriography, but stated that large tumor size might be simply related to referral bias as those

are the most likely meningiomas to be embolized. No association with meningioma location or histologic subtype was found. Kallmes et al also pointed out that while smaller diameter GF powder and 50 to 150 μ m PVA was used in four of seven cases, hemorrhages were also seen with intermediate size particles. More recently, Bendszus et al reported hemorrhage in 3.2% (5/185) of patients treated with ES (100–300 μ m and 40–120 μ m). Patients presented within 24 hours of embolization with headache ($n = 3$), hemiparesis ($n = 2$), or coma ($n = 1$). Rapid surgical treatment led to neurological recovery in four patients, but the other patients expired (0.5% mortality). MRIs and arteriograms were not different from those in patients who did not experience postembolization hemorrhage; however, histological examination revealed atypical subtype (40%), pathological dilated thin-walled vessels (80%), and signs of previous hemorrhage (60%). The authors suggested a close review of preangiography MRI for signs of prior bleeding.

Ischemic complications occur in less than 3% of cases and are very often transient.^{20,24} However, they may lead to permanent CN palsies,²⁴ blindness,³ hemiparesis,²¹ and even death.²¹ The jaw pain and trismus described with IMAX/MMA embolization is related to unintended embolization of branches to jaw muscles and generally is self-limited.⁹ Ischemic complications to CNs are important to consider, especially when embolizing the MMA given its supply to the seventh CN (petrosal branch, which may be the dominant supply to the facial nerve in 25%), as well as other collaterals. Berenstein and Kricheff⁵ recommended microcatheter placement 15 mm beyond the foramen spinosum or the use of larger particles to reduce the chance of this complication. Furthermore, since the vasa nervorum are generally less than

150 μm , several authors have suggested not using particles smaller than this to help reduce the risk of CN injury.^{7,8} When one is uncertain, peripheral nerve testing may be performed with 20 mg of intra-arterial lidocaine.^{7,55} Thromboembolic complications are not completely avoidable, but will generally be reduced by using meticulous technique and judicious anticoagulation.

Conclusions

Meningioma embolization has been performed successfully for 40 years. Contemporary angiographic imaging and neurovascular equipment have enabled visualization and catheterization of the very distal vasculature with a 2 to 6% neurological complication rate in most modern series. Discussion persists regarding the indications for embolization and its efficacy. Current evidence suggests that preoperative embolization can reduce intraoperative blood loss, reduce transfusion requirements, and make surgery easier in appropriately selected cases. It remains unknown whether embolization reduces meningioma recurrence rates after surgery. As with all interventions, maximal patient benefit requires careful patient selection and preparation (i.e., steroids, meticulous technique, and good judgment). Appropriate patient selection is performed in a multidisciplinary fashion and depends on the neurosurgeon's preference, tumor location, tumor blood supply, surgical risk assessment, and the IRS experience.

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