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Review Article

Transarterial Embolization of a Parasellar Hypervascular Tumor

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Embolization of hypervascular tumors has been widely performed for over four decades, particularly for preoperative meningioma. Several benefits of preoperative embolization have been reported, including reduced blood loss, surgical time and surgical complications, and improved outcomes. However, the technical details of both embolization and surgical procedures, and lesions widely vary. Thus, the actual benefits of preoperative embolization have not been clarified by prospective randomized studies. Procedure-related complications due to embolization developed in 3%–12% in previous studies. For parasellar lesions, both surgical resection and embolization have a higher risk of complication than for lesions at other locations because of the complicated neurovascular anatomy in the parasellar area. Therefore, close attention should be paid to the detailed vascular anatomy, embolic material, and related information for embolization and resection in individual cases to improve patient outcomes.

Keywords ▶ parasellar tumor, meningioma, embolization

Introduction

Surgical resection of parasellar hypervascular tumors, such as petroclival meningioma, is challenging because of the deep and narrow surgical field, and deep vascular supply to the tumor.[1)] Although early operative devascularization is one of the key points in surgical resection of hypervascular tumors, some deep feeders may be difficult to devascularize until the later stages of tumor resection. Preoperative embolization has been widely used for these hypervascular tumors to reduce intraoperative blood loss, surgical time, and perioperative complications, and to promote the complete resection of tumors.[2,3)] However, transarterial embolization has a potential risk of complications, including tumor bleeding, cranial nerve injury, and stroke due to the migration of embolic material. Parasellar lesions in particular have a higher risk of complications due to the complicated neurovascular anatomy.[4)] Therefore, it is important to know the functional vascular anatomy of the parasellar area and techniques of embolization. We report important issues for transarterial embolization of parasellar hypervascular tumors, including arterial anatomy, embolic materials, and other factors associated with clinical outcomes.

Arterial Anatomy

As described in "arterial anatomy," the parasellar area contains a complicated arterial network, including anastomosis between external carotid arterial branches and branches from the internal carotid artery (ICA) and/or ophthalmic artery (OPA), and parasellar tumors can be fed by all of these arterial systems. The inferolateral trunk (ILT) and meningohypophyseal trunk (MHT) from the ICA, and the recurrent meningeal artery (superficial recurrent OPA) and deep recurrent OPA from the OPA provide feeders to parasellar hypervascular tumors **(Figs. 1** and **2).**[5)] In cases supplied by branches of both the external carotid artery (ECA) and ICA or OPA with anastomosis, embolic material injected from ECA branches can migrate into the ICA or OPA, which may cause serious complications such as stroke or blindness.[4)] These branches from the ICA and

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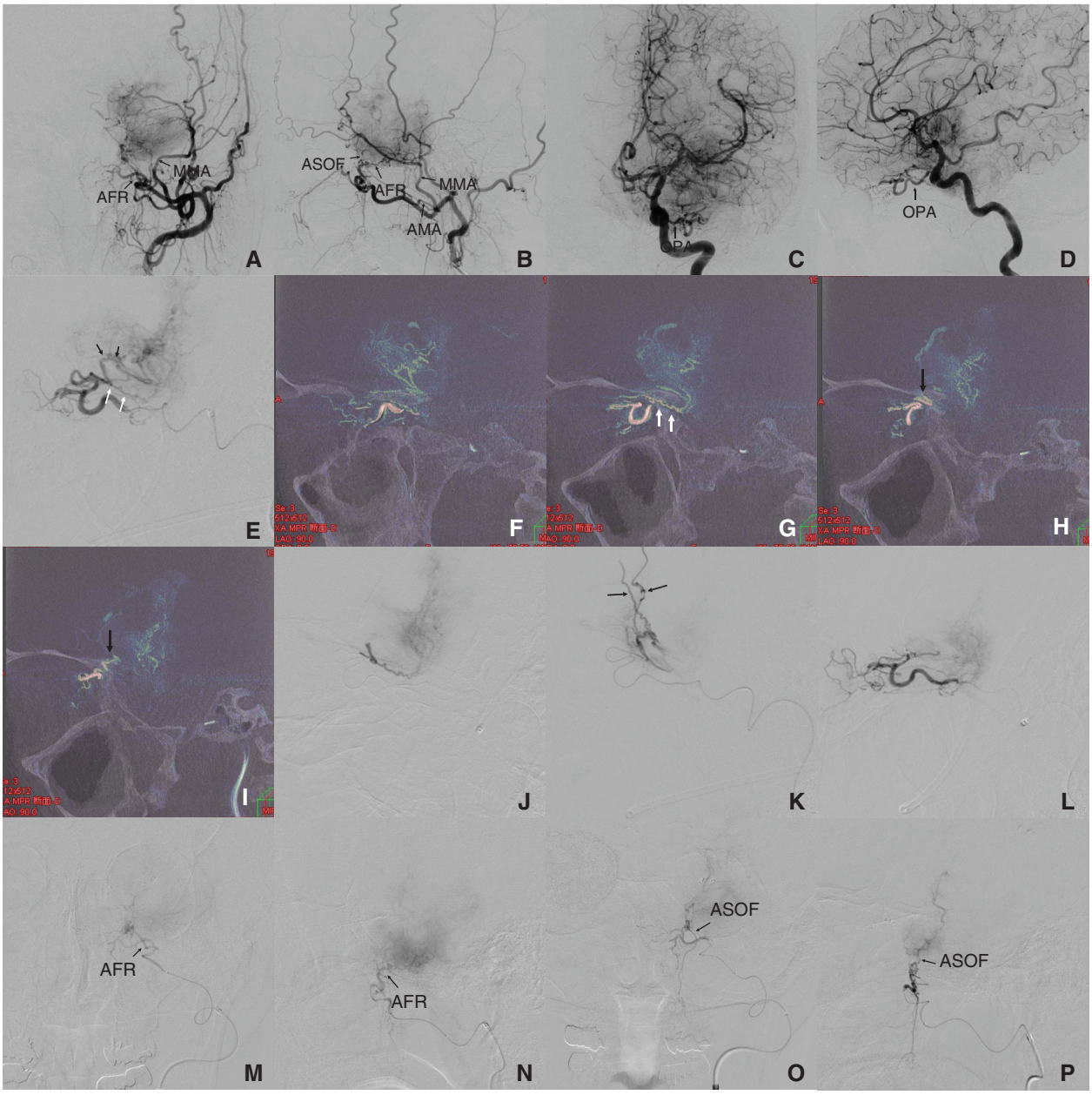

Fig. 1 Left sphenoid ridge meningioma fed by the multiple feeders from the ECA and OPA. Frontal (**A**) and lateral (**B**) views on left external carotid angiography show a large contrast blush supplied by numerous feeders from the MMA, AMA, AFR and ASOF. Frontal (**C**) and lateral (**D**) views on left internal carotid angiography show the mass supplied by feeders from the OPA. The distal portion of the left ICA, and proximal portion of the middle cerebral and anterior cerebral arteries are displaced by the mass. (**E–I**) Lateral view (**E**) and sagittal MPR images (**F–I**) on left ophthalmic angiography show the mass being fed by the recurrent meningeal artery (superficial recurrent OPA, black arrows) and deep recurrent OPA (white arrows). The recurrent meningeal artery runs through the superolateral part of the superior orbital fissure into the cranial cavity, and may anastomose with the anterior branch of the MMA. The deep recurrent OPA runs through inferomedial portion of the superior orbital fissure and may anastomose with the artery of the superior orbital fissure and ILT. (**J**) Lateral view on selective angiography of the deep recurrent OPA shows contrast blush at the anterior part of the mass. Feeders from the deep recurrent OPA and recurrent meningeal artery were embolized using small gelatin sponge pieces. (**K**) Lateral view on selective angiography after embolization of feeders from the recurrent ophthalmic arteries shows opacification of the MMA (arrows) via meningo-lacrymal anastomosis. Two coils were next placed via the microcatheter. (**L**) Lateral view on left ophthalmic angiography immediately after embolization shows marked devascularization. Small feeders from the first segment still remain. Frontal (**M**) and lateral (**N**) views on maxillary angiography show contrast blush fed mainly by the AFR. Frontal (**O**) and lateral (**P**) views on maxillary angiography after embolization of the AFR show residual contrast blush fed by the ASOF. The ASOF was also embolized using particles. AFR: artery of foramen rotundum; AMA: accessory meningeal artery; ASOF: artery of the superior orbital fissure; ECA: external carotid artery; ILT: inferolateral trunk; MMA: middle meningeal artery; MPR: multi-planar reformatted; OPA: ophthalmic artery

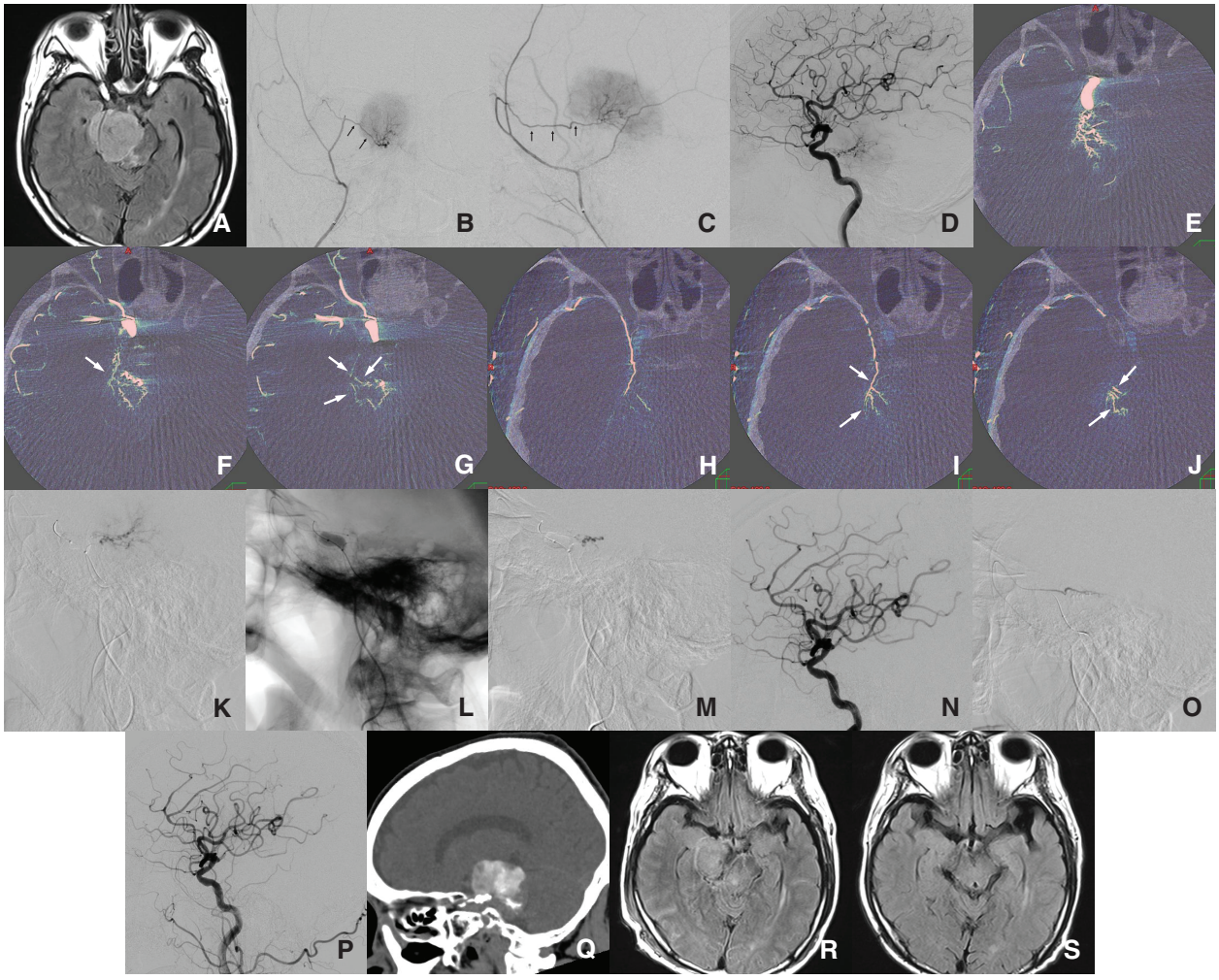

Fig. 2 Petroclival meningioma fed by feeders from the meningohypophyseal trunk and the MMA. (**A**) FLAIR MR image shows a hyper-intense mass at the petroclival region. The mid-brain is displaced by the mass. Frontal (**B**) and lateral (**C**) images on right middle meningeal angiography show contrast blush fed by feeders from the anterior branch (recurrent meningeal artery, arrows) (**D**) Lateral view of the right internal carotid angiography showing the feeders from the meningohypopyseal trunk supplying the mass. (**E–J**) Axial MPR images on right internal carotid angiography (**E–G**) and external carotid angiography (**H–J**) show the feeders (white arrows) from the anterior branch of the MMA, which were also opacified from the meningohypophyseal trunk of the right internal carotid artery. Therefore, there is a potential risk of migration of embolic material injected from the MMA into the internal carotid artery. (**K**) Lateral view on selective angiography via a 1.3-F microcatheter advanced into the MHT trunk shows feeders supplying the tumor. (**L**) Lateral fluoroscopic image during embolization shows a microcatheter in the meningohypophyseal trunk and a balloon catheter was placed crossing over the orifice of the meningohypopyseal trunk. (**M**) A 20% NBCA–Lipiodol mixture was injected via the microcatheter into the meningohypopyseal trunk. (**N**) Lateral view on right internal carotid angiography after embolization of the meningohypophyseal trunk shows the disappearance of contrast blush and feeders. (**O**) Lateral view of selective injection of the 20% NBCA–Lipiodol mixture. A residual feeder of the anterior branch of the MMA was embolized using the 20% NBCA–Lipiodol mixture. (**P**) Lateral image on right common carotid angiography shows complete devascularization. (**Q**) Sagittal image of brain CT immediately after embolization shows cast of NBCA-Lipiodol mixture and retention of contrast media in the tumor. Planned surgical resection was performed one day after embolization. (**R**) FLAIR MR imaging after surgical resection shows a residual mass compressing the mid-brain. (**S**) FLAIR MR imaging 1 year after surgical resection showing marked regression of the residual mass. MHT: meningohypophyseal; NBCA: N-butyl cyanoacrylate

OPA can often be catheterized.[6] Therefore, embolization of the feeders from the ICA or OPA before embolization of ECA feeders can prevent the migration of embolic material into the ICA or OPA (**Figs. 1** and **2**).

Among the branches of the ECA, the middle meningeal artery (MMA) often provides main feeders to parasellar tumors, which include cavernous sinus branches, the petrosal branch, petrosquamosal branch, and anterior branch.[7] The cavernous sinus branch and petrosal branch originate from the proximal portion of the horizontal segment of the MMA, and run tortuously to supply the tumor. The petrosal branch is an important artery because it has potential anastomoses with the tentorial arteries of the ILT or MHT, and anterior inferior cerebellar artery. Furthermore, a petrosal branch, the superficial petrosal artery, runs through the facial nerve canal and anastomoses with the stylomastoid

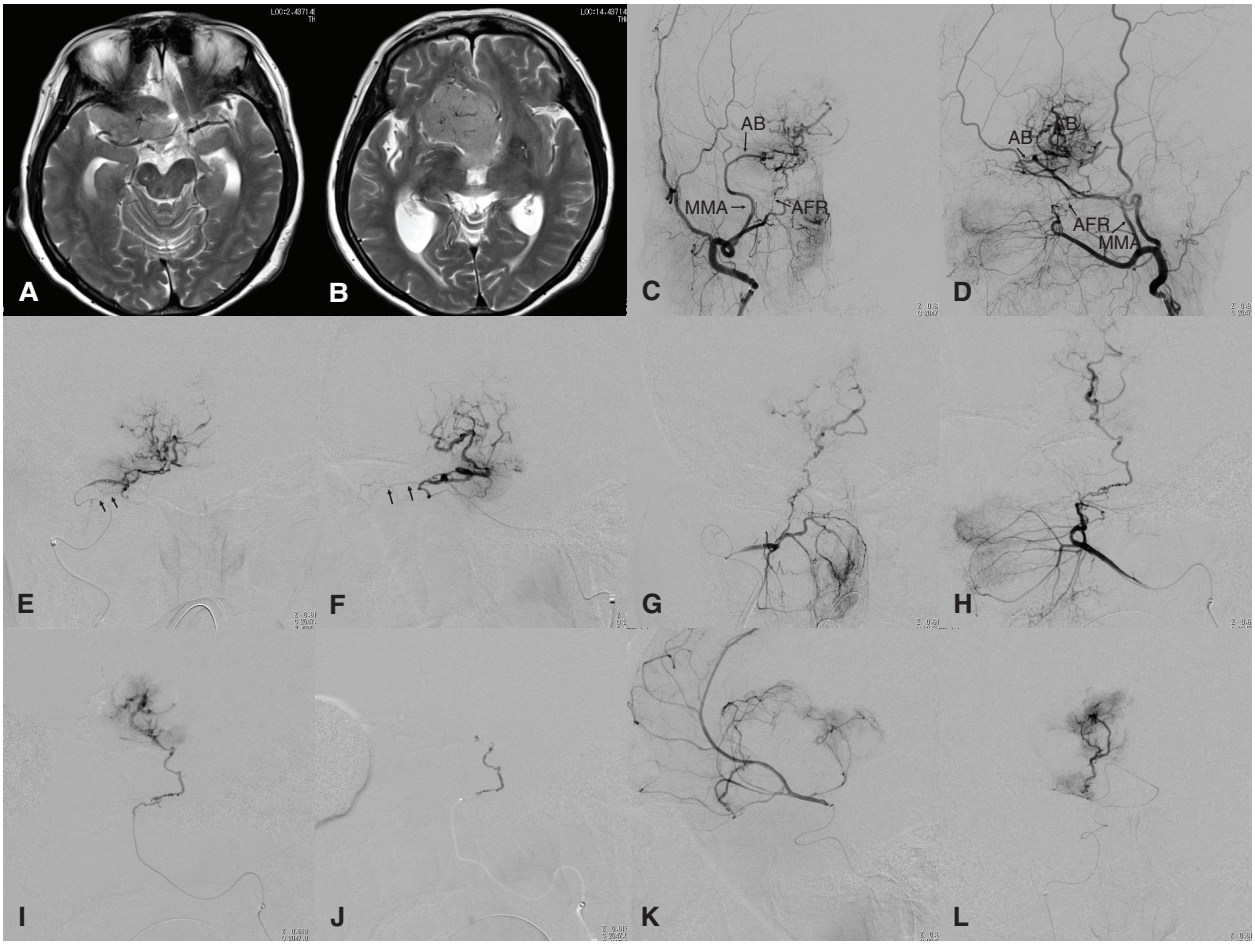

Fig. 3 A large sphenoid ridge meningioma with pial arterial supply. (**A** and **B**) T2-weighted MR images show a large mass at the right sphenoid ridge growing upward and displacing the right frontal lobe. Frontal (**C**) and lateral (**D**) views on right external carotid angiography show strong contrast blush and markedly hypertrophied feeders from the AntB of the MMA and the AFR. Frontal (**E**) and lateral (**F**) views on selective angiography of the anterior branch of the MMA show marked dilated tumor vessels. An orbital branch (meningolachrymal artery, arrows) is noted. The anterior branch was embolized using large particles (300–500 Embosphere) and gelatin sponge pieces. Frontal (**G**) and lateral (**H**) views on selective maxillary angiography at the third portion show the AFR supplying the tumor. (**I**) Lateral view on selective angiography of the AFR during embolization using microspheres shows further dilatation of the tumor vessels despite reducing contrast blush. (**J**) Due to the potential risk of tumor bleeding, the 20% NBCA–Lipiodol mixture was injected via the AFR to occlude the feeder. Selective angiography of the anterior cerebral artery (**K**) and the feeder from the anterior cerebral artery (**L**) show that the superomedial part of the tumor was supplied from pial feeders. The feeder was embolized using the NBCA–lipiodol mixture. AFR: artery of foramen rotundum; AntB: anterior branch; MMA: middle meningeal artery; NBCA: N-butyl cyanoacrylate

artery while supplying the facial nerve. Therefore, non-target embolization with small particles or liquid material from the petrosal branch carries a risk of ischemic stroke and facial nerve injury. The petrosquamosal branch originates posteriorly from the distal portion of the horizontal segment, and runs posteriorly along the petrosquamosal fissure to supply feeders running medially on the posterosuperior surface of the petrous bone to parasellar tumors at a petroclival location. Embolization from the petrosquamosal branch is relatively safe but it also has potential anastomosis with the ICA via tentorial arteries. The anterior branches originate from the distal portion of the temporal segment. They run medially along the lower surface of the sphenoid ridge, and can supply the tumor at the sphenoid ridge and cavernous sinus. As it frequently anastomoses with the lachrymal branch of the OPA, careful attention should be paid to migration of embolic material to the OPA.[4] Other maxillary arterial branches supplying the parasellar tumor include the accessory meningeal artery, artery of foramen rotundum, and artery of superior orbital fissure (**Figs. 2** and **3**). They all have branches of vasa nervosa and potential anastomoses with the corresponding branch of the ILT. Furthermore, the artery of the superior orbital fissure can anastomose with the OPA.[8] Petroclival lesions can be supplied by the medial and lateral clival arteries from the ascending pharyngeal artery, which can anastomose with

the dorsal clival artery of the meningohypopyseal trunk. Large tumors are often fed by pial feeders from the middle cerebral artery or the anterior cerebral artery (**Fig. 3**). These pial feeders may have peritumoral anastomoses with dural feeders from the ECA. The recent development of microcatheter technology has enabled us to perform embolization from pial feeders.[9] However, catheterization and embolization from pial feeders have potential periprocedual risks; therefore, its application is assessed on a case-by-case basis.

Knowledge of the detailed vascular anatomy described above is essential to avoid serious complications.

Embolic Materials

Many embolic materials, including several types of particles, powders, liquids, and coils, have been used for transarterial embolization of intracranial or skull base hypervascular tumors. Each embolic material has some advantages and disadvantages, and should therefore be selected for each case and target artery.

Particles

Historically, absorbable gelatin sponge particles or powder were used, but were mostly replaced by polyvinyl alcohol (PVA) particles and microspheres.[10,11] PVA particles are considered a nonbiodegradable and permanent embolic agent. They mechanically occlude the target vessel, but can lead to inflammatory reaction and thrombosis. PVA particles are prepared as irregular particles with varying standardized size ranges. There is variability in their size beyond the standardized size range because of their irregular shape. Smaller or larger particles may lead to unintended distal or proximal embolization. PVA particles potentially expand when they contact water and they usually aggregate together when suspended in saline. Therefore, they often occlude blood vessels slightly larger than that estimated by the size of dried particles. There is a potential risk of occlusion of the microcatheter lumen by aggregation of PVA particles during delivery, necessitating frequent flushing of the microcatheter.

Microspheres, spherical embolic materials, have been developed to overcome some of the disadvantages of PVA particles, including size variability in a given preparation of particles and particle aggregation. Since their introduction, microspheres have been employed worldwide, particularly for embolization of hypervascular tumors in numerous organs. Microspheres have a more reliable size range. They are more deformable, and do not aggregate due to their spherical shape and hydrophilic nature. These characteristics reduce the risk of proximal occlusion and microcatheter occlusion during delivery. Several types of microspheres have been commercialized, including trisacryl gelatin microspheres (Embosphere, Nipponkayaku, Tokyo, Japan), polyphosphazene-coated polymethylmethacrylate (PMMA) microspheres (Embozene, Boston Scitentific, Marlborough, MA, USA), and acrylamido PVA microspheres (Bead Block, Boston Scitentific, Marlborough, MA, USA). These microspheres demonstrate different compressibility and deformability, which affect the embolized vessel. Bendszus et al. reported the superiority of 100- to 300-μm tris-acryl spheres (embosphere) to both small-sized (45–150 μm) and medium-sized (150–250 μm) PVA particles.[12] According to their study, intraoperative blood loss was significantly lower in the group using tris-acryl spheres than in both PVA groups (621 vs. 881 vs. 917 mL: $p < 0.05$). Histological specimens revealed that the microspheres penetrated significantly more distally into the tumor than PVA particles ($p < 0.005$). In addition, small-sized PVA particles exhibited more distal penetration than medium-sized PVA particles. Smaller sized (<150 μm) PVA particles and microspheres can penetrate deeply in the tumor vessels, which may be more effective to induce tumor necrosis than larger particles.[13] However, they have a higher risk of complications due to ischemia of the normal tissue, cranial nerve injury, and hemorrhage. Rosen et al. reported the efficacy and complications of preoperative embolization using 50-μm PVA particles for hypervascular skull base tumors in 167 patients. In their study, transient and permanent neurological deficits, including stroke, cranial nerve deficits, and tumor bleeding, were found in 21 patients (12.6%) and 15 patients (9%), respectively.[4] Tumor bleeding during or immediately after embolization may be caused by blockage of the drainage vein by small particles due to their deep penetration into the drainage vein.[14] It is well known that intratumoral extravasation, the so-called "vascular lake," often develops during embolization of hepatocellular carcinoma using microspheres (**Fig. 4**).[15] The risk of hemorrhagic complications by microspheres may be higher than that of the same sized PVA particles because microspheres can penetrate deeper (**Fig. 4**).[16]

Liquid

N-butyl cyanoacrylate (NBCA) and ethylene-vinyl alcohol copolymers, such as ONYX, have recently been used for the embolization of hypervascular tumors. NBCA is a liquid adhesive and one of the most common liquid embolic materials. NBCA polymerizes upon contacting anions in the

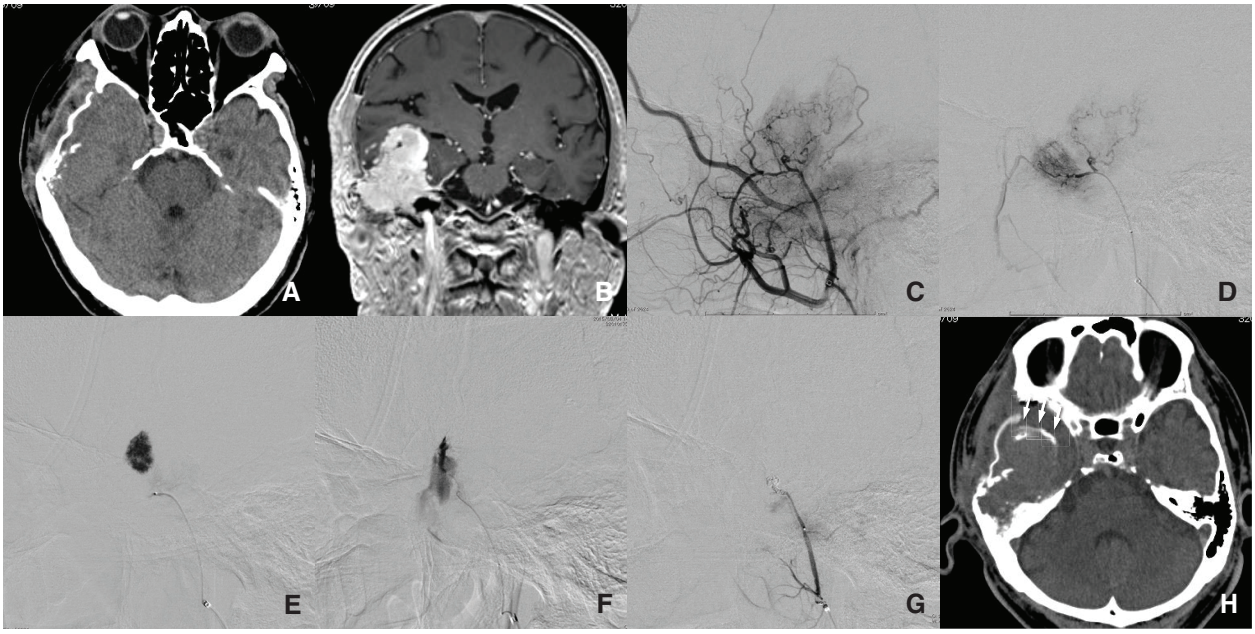

Fig. 4 Recurrent meningioma at the middle cranial fossa. (**A**) Brain CT shows a large iso-density mass at the right middle fossa. Bone defect of the right temporal bone due to previous surgery was noted. (**B**) Coronal image of post-contrast T_1-weighted MRI shows a markedly enhanced mass at the right middle cranial fossa. The mass invaded into the infratemporal fossa with destruction of the skull base. (**C**) Lateral view of the left external carotid angiography shows the interruption of the right MMA due to previous surgery and numerous small feeders originating from the proximal portion of the MMA, accessory meningeal artery, and deep middle temporal artery. (**D**) Selective angiography of a feeder from the MMA shows strong contrast blush with early venous drainage. (**E**) Selective angiography during embolization unsung microspheres (300–500 microsphere) shows contrast pooling in the tumor, termed "vascular lake." (**F**) Repeated selective angiography a few minutes later shows extravasation. The feeder was immediately embolized using coils. (**G**) Middle meningeal angiography after embolization shows the disappearance of the extravasation. Marked regression of tumor staining was also noted. (**H**) CT immediately after embolization shows contrast leakage in and around the tumor (white arrows). MMA: middle meningeal artery

blood when injected into the vessel, after which it becomes adhesive and occludes vessels instantly.[17] NBCA is mixed with an iodized oil, such as Lipiodol (Lipiodol, Guerbet Japan, Tokyo, Japan), to make the embolic agent radiopaque and to prolong the polymerization time. It has been frequently used for embolization of arteriovenous shunts, and recently become commonly employed for the embolization of hypervascular tumors such as meningioma.[18–21]

More recently, Onyx has been applied for the embolization of intracranial hypervascular tumors.[22] The non-adhesive nature of ONYX enables prolonged injection and deep penetration in the tumor vessels. A small case series reported good results of embolization using ONYX for meningiomas.[23] However, the superiority of using ONYX to other materials for embolization of hypervascular tumors has not been clarified because no large cohort study has been published.

Utility of Preoperative Embolization

Transarterial embolization of intracranial hypervascular tumors has been performed for over four decades as an adjunctive technique to surgical resection to reduce intraoperative blood loss, operative duration, and surgical morbidity, and increase the resection rate. Several studies demonstrated the utility of preoperative embolization compared with surgical results without embolization.[3,13,20] In addition, preoperative embolization is associated with a longer progression-free period.[3] However, the benefits of preoperative embolization remain controversial.[11,24] Rapper et al. retrospectively compared the operative duration, degree of resection, surgical blood loss, and surgical complications between two groups with and without preoperative embolization using PVA particles in 470 patients with 504 meningiomas. However, there were no significant differences in operative duration, degree of resection, or surgical complications between the two groups. Independent factors for operative blood loss in the multivariate analysis included skull base location (71% are in parasellar areas) and incomplete embolization.[25] As described above, parasellar tumors have a complicated vascular supply, which is often a dual supply from branches of the external and internal carotid arteries. Furthermore, these feeders often originate from neuromeningeal arteries, which extend small branches to supply cranial nerves. Therefore, aggressive embolization of parasellar lesions has a higher risk of neurological complications, but sufficient embolization is

required to be a beneficial adjunct to surgery. On the other hand, the outcomes of resection of parasellar hypervascular tumors, such as anterior clinoid meningioma, without preoperative embolization reported were gross or total resection rates of 30%–80%, neurological complication rates of 10%–50%, 0%–4% mortality, and a recurrence rate of 10%–50%.[1,26–29] To our knowledge, no studies regarding embolization of parasellar tumors have been reported. Ilyas et al. recently reported a systematic review of preoperative embolization for skull base meningiomas, among which 77% were located at the parasellar region.[30] They reviewed 403 cases from 15 studies, and reported gross or total resection rates of 74%, a major complication rate of 12% and 0.2% mortality. They concluded that preoperative embolization is a reasonable adjunct to resection for appropriately selected skull base meningiomas.

Timing of Surgery after Embolization

The time interval between presurgical embolization and surgery is a matter of debate. Chun et al.[31] retrospectively reviewed meningioma patients who underwent preoperative embolization and surgical resection within 24 h or after 24 h, and they observed significantly less blood loss in the group with surgical resection 24 h after embolization. Nania et al.[13] retrospectively compared the surgery time and transfused blood volume among the three groups (surgery performed less than 7 days after embolization, at least 7 days after embolization, and no embolization). In their study, the surgery time and transfused blood volume were significantly lower in the group that underwent surgery at least 7 days after embolization. They also noted that large confluent areas of necrosis were significantly more frequent in patients with a larger time span between embolization and surgery. Kai et al.[32] investigated 42 cases of embolized meningioma, and demonstrated that the greatest tumor softening occurred 7–9 days after embolization and that the optimal interval between embolization and surgery was 7–9 days. In contrast, several authors recommended surgery after embolization within 7 days due to tumor revascularization from recanalized feeders and/or development of collateral feeders. However, recanalization and collateralization may be rare within 2 weeks if the intratumor vessels are occluded with permanent embolic materials. Another concern is the development of edema after embolization. The deterioration of symptoms due to aggravation of edema may require immediate surgery after embolization. Therefore, the optimal time interval may be 7–9 days after embolization except for specific cases have a high risk of aggravated edema after embolization.

Definitive Embolization

The possibility of embolization as a sole therapy for benign hypervascular tumors was discussed in a few case reports.[33] Bendszus et al. treated seven cases of meningioma by embolization alone using trisacryl gelatin microspheres.[33] Complete angiographic devascularization was obtained in five patients and partial devascularization in two patients, and six exhibited continuous tumor shrinkage and improvement of symptoms during the mean 20-month follow-up period.[34] Although further investigation using a larger cohort is needed, it may be an alternative option for the treatment of parasellar hypervascular tumors in selected patients with high surgical risk.

Summary

Embolization of hypervascular tumors is a classic technique and beginners in neuroendovascular treatment may often be involved in this field. However, the required skills and knowledge for safe and effective embolization of parasellar lesions are different from those for lesions in the convexity or parasagittal areas. Regarding this area, how to safely and sufficiently embolize dangerous feeders in individual cases is important.

Disclosure Statement

The authors declare no conflict of interest.

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