Preoperative Endovascular Embolization of Intracranial Meningiomas

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Introduction

Meningiomas are the most common primary brain tumor. Although typically benign, meningiomas may cause seizures, neurological deficits, or vision loss through mass effect on the surrounding brain parenchyma. Surgical resection is the definitive treatment in such cases. Meningiomas have a propensity to parasitize arterial blood supply from the extracranial or intracranial circulation, and blood loss reported up to 2L is a significant perioperative risk. [1,2] Preoperative endovascular embolization of meningiomas is an important adjunct therapy that reduces blood loss and operation time.[3,4]

The purpose is to report on technical considerations involved in preoperative embolization of intracranial meningiomas. We present four cases to highlight the associated vascular anatomy, with a focus on identifying dangerous anatomic pathways to avoid complications related to non-target embolization.

Methods

The same neurointerventional radiologist performed all four procedures. The common carotid arteries, internal carotid arteries, and external carotid arteries (ECA) were interrogated with particular attention to the superficial temporal artery (STA) and middle meningeal artery (MMA). Excelsior SL-10 (Stryker, Salt Lake City, UT) and Excelsior XT-27 microcatheters (Stryker, Salty Lake City, UT) were utilized in combination with Synchro .014'' microwires (Stryker, Salt Lake City, UT) to identify dural feeders and avoid dangerous anastomotic pathways. Embolization was performed with either 150-250 micron, 300 micron, or 500 micron Embosphere particles (Merit Medical, South Jordan, UT). Images were reviewed on Centricity PACS (GE Healthcare, Chicago, IL). Neurosurgical outcomes were reviewed in Meditech Electronic Health Records (Meditech, Westwood, MA). All patient information was handled in compliance with HIPAA.

Dangerous Anastomotic Pathways

Extracranial		Intracranial	
Major Artery	**Branch Artery**	**Branch Artery**	**Artery**
IMAX	MMA	Orbital branch	Ophthalmic artery
		Cavernous branch	Inferolateral trunk
		Petrous branch	CN VII supply
	AMA	Artery of foramen ovale	Inferolateral trunk
	Vidian artery		Petrous ICA
	Anterior deep temporal artery		Ophthalmic artery
STA		Supraorbital branch	Ophthalmic artery
Ascending pharyngeal	Superior pharyngeal	Carotid branch	Lateral clival artery
	Odontoid arch		Vertebral artery (C1)
	Hypoglossal and jugular branch		Meningohypophyseal trunk
Posterior auricular-occipital	Stylomastoid branch		CN VII supply
Occipital artery	Muscular branches		Vertebral artery (C1-2)
Ascending, deep cervical			Vertebral artery (C3-7)
IMAX: internal maxillary, AMA: accessory meningeal, STA: superficial temporal, MMA: middle meningeal			

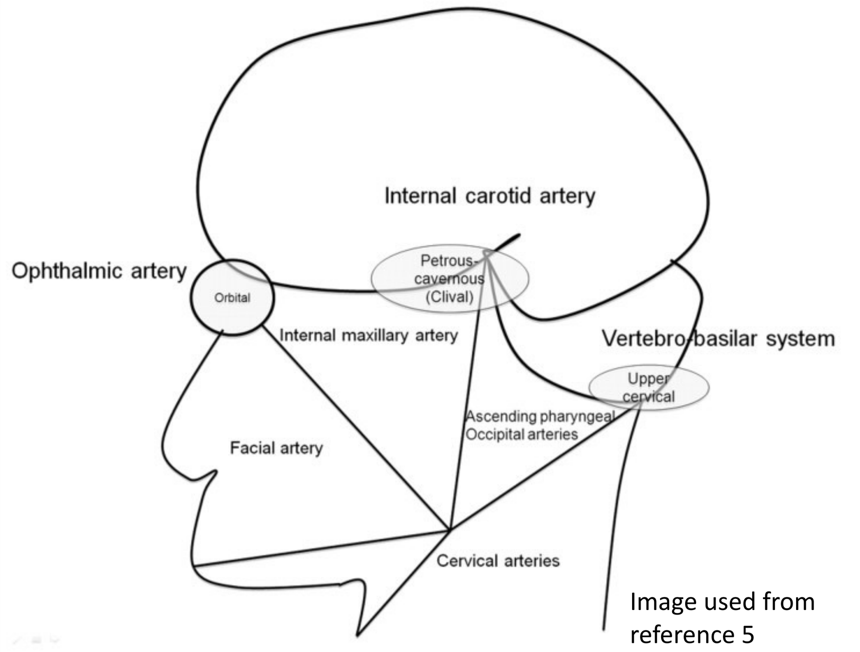
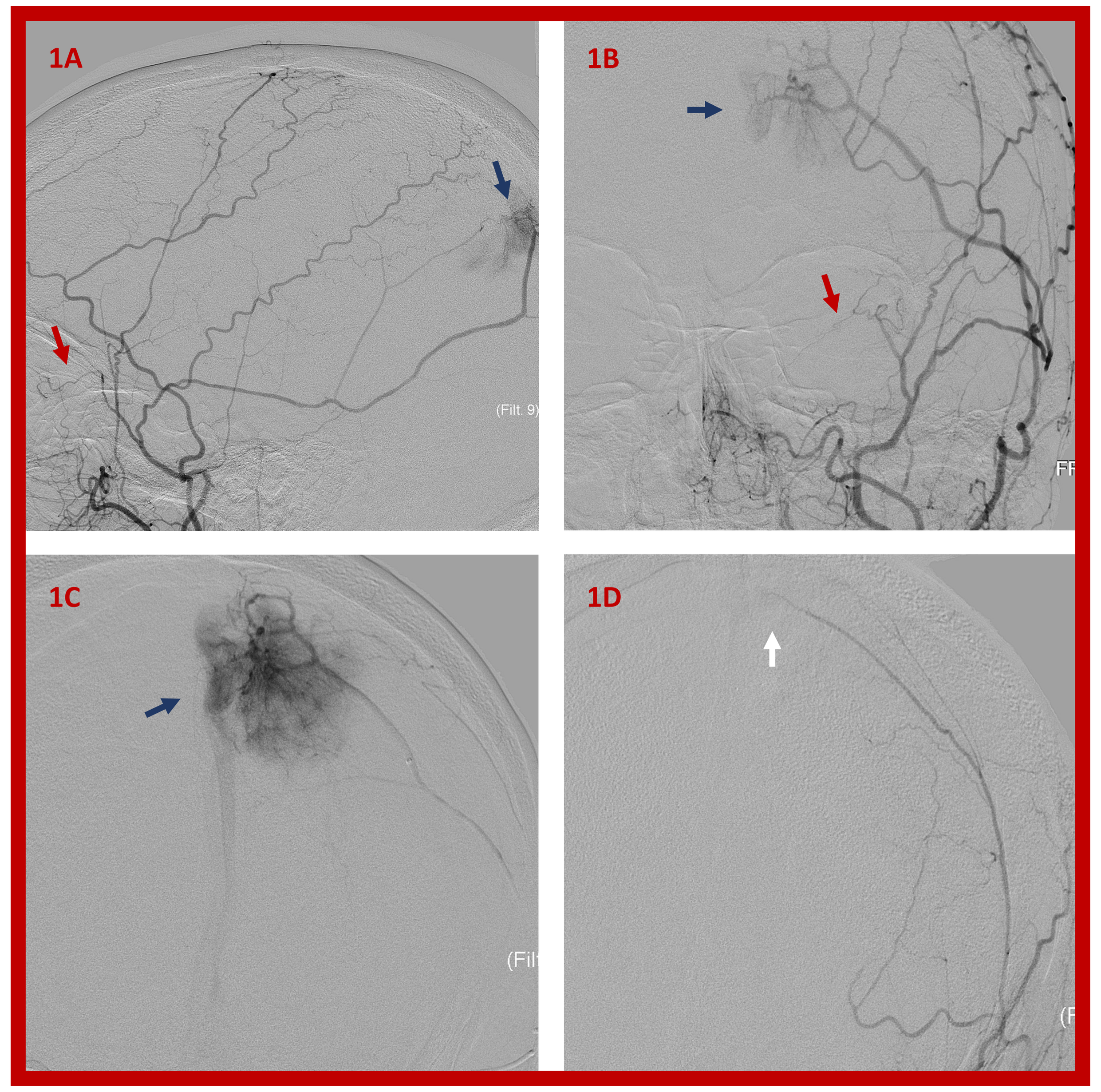
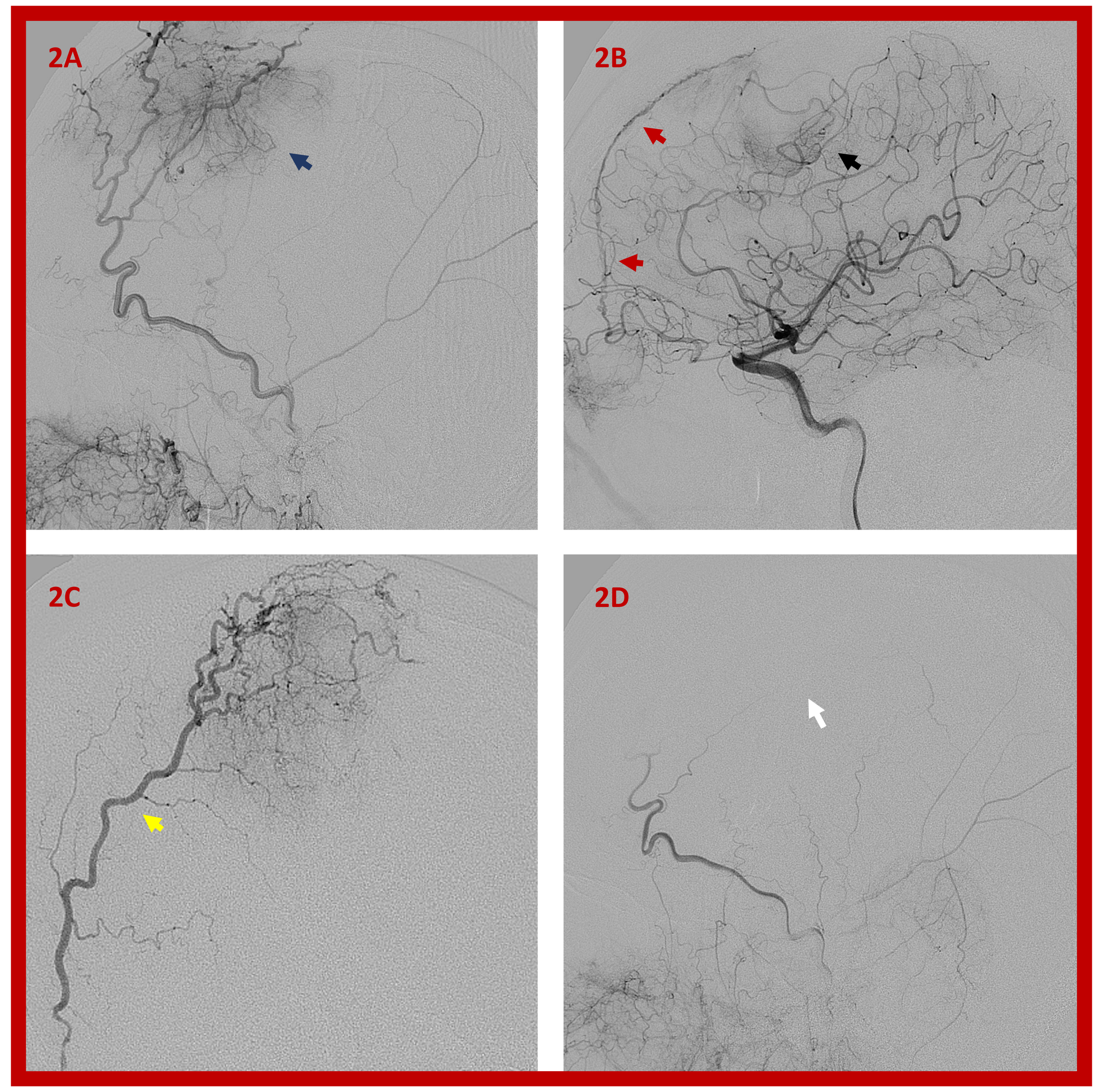


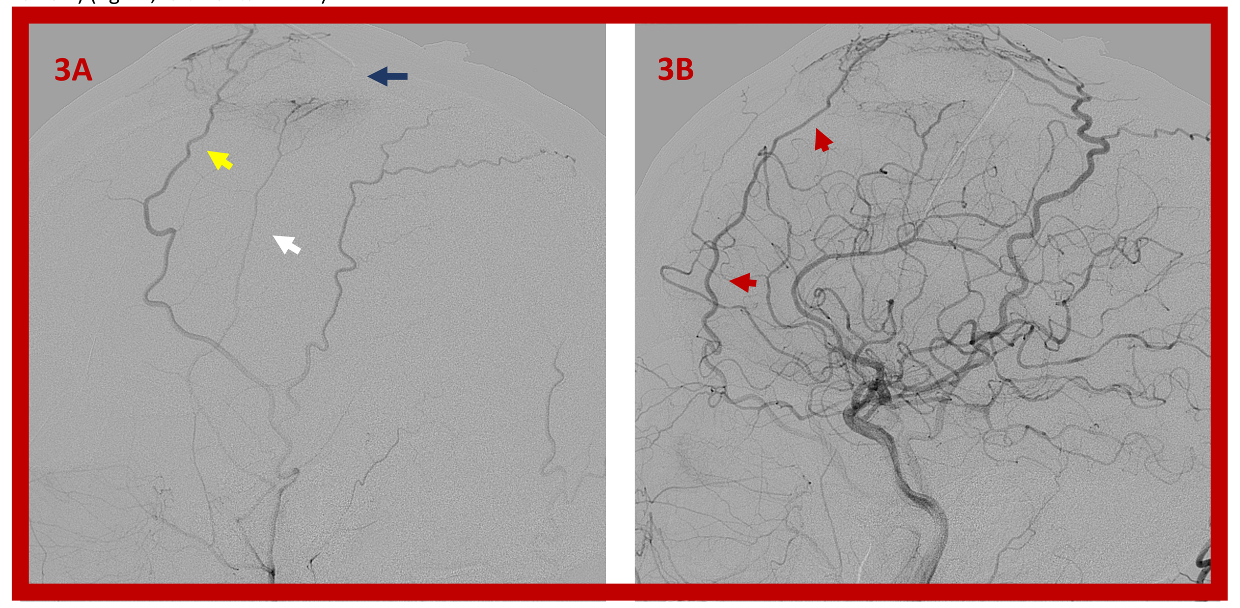
Image used from reference 5



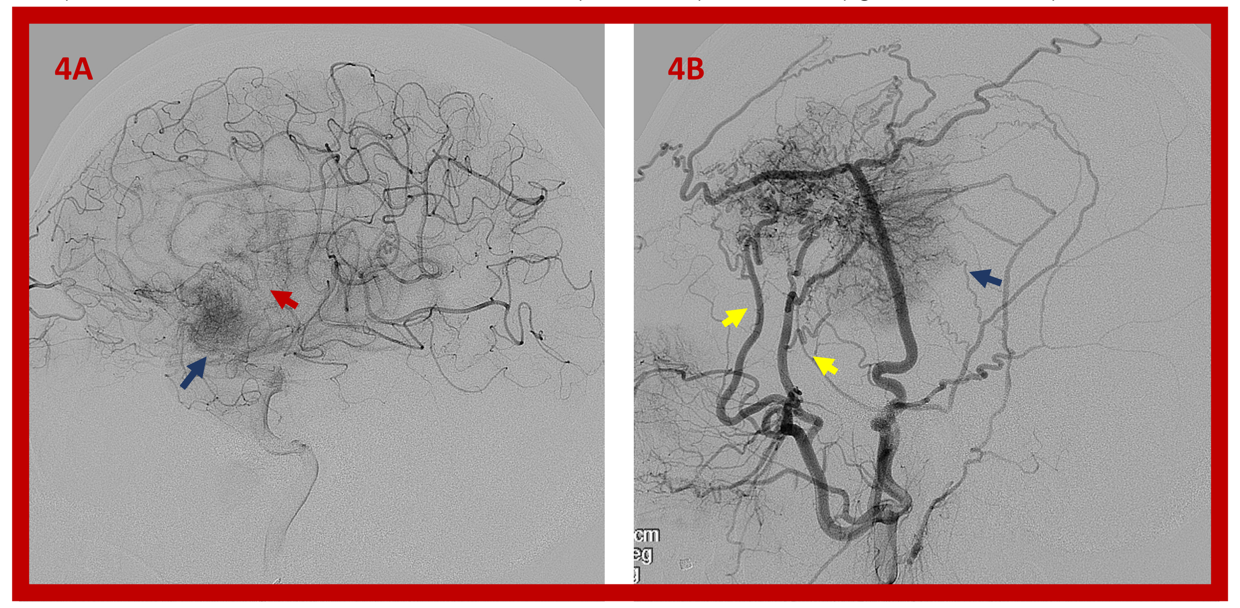
Left parasagittal meningioma (blue arrows) fed via the middle meningeal artery, with a small transorbital branch (red arrow) identified arising from the MMA and coursing medially (fig 1A, left lateral ECA, fig 1B, left frontal ECA). The microcatheter was advanced past this vessel (fig 1C, left frontal MMA). Post embolization shows successful resolution of tumor blush (white arrow) (fig 1D, left frontal MMA).



Left frontal meningioma (blue arrow) arising from STA, MMA branches (fig 2A, left lateral ECA), as well as branches from the right STA (yellow arrow) (2C, right frontal ECA). Significant left intracranial supply via the anterior falcine artery arising from the ethmoidal branch of the ophthalmic artery (red arrows) and intracranial ACA branches (black arrow) (fig 2B, left lateral ICA). Post embolization reveals resolution of tumor blush (white arrow) from the ECA (fig 2D, left lateral ECA).



Left frontal meningioma (blue arrow) fed via STA (yellow arrow) and MMA (white arrow) branches (fig 3A, left lateral ECA). Minimal supply noted from left anterior falcine artery (red arrow) arising from the ophthalmic artery. (fig 3B, left lateral CCA)



Right frontotemporal meningioma (blue arrows) with intracranial supply via the right MCA branches (red arrow) (fig 4A, right lateral ICA). Hypertrophied STA (yellow arrow) and MMA are the main source of meningioma supply. (fig 4B, right lateral ECA)

Results

Cerebral angiography revealed meningiomas supplied from branches of the ECAs, STAs, and MMAs. The interventional endpoint of stagnant tumor flow was achieved in each case. All patients underwent successful neurosurgical resection. Estimated intraoperative blood loss ranged from 100 to 200 cc. Postoperative hemoglobin decreases of 1.1 g/dL, 1.2 g/dL, 1.3 g/dL, and 2.0 g/dL were reported. No complications occurred in the described patient cohort.

Discussion and Conclusion

Reported success rates are between 91-100%.[6] The ideal interventional endpoint is a lack of contrast blush within the tumor, however complete embolization is oftentimes limited due to multiple dural feeders containing dangerous anastomotic pathways and supply from intracranial arteries. Non-target embolization may cause unintended ischemia, scalp necrosis, and various cranial nerve palsies. The use of particles and an understanding of these anatomical pathways is critical to avoiding devastating consequences related to non-target embolization. Preoperative embolization of intracranial meningiomas is an important adjunct therapy that helps limit blood loss and reduce perioperative risk.

Selected References

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