Pre-operative Embolization of Intracranial and Extracranial Tumors: A Review of 37 Cases

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Objective: To examine the safety and efficacy of pre-operative embolization of intra-and extracranial tumors and determine the selection criteria of patients for this procedure.

Material and Method: Between June 2008 and August 2012, 37 patients (17 males, 20 females; mean age, 44.2±14.2years), underwent pre-operative embolization of intra- and extracranial tumors, were retrospectively reviewed. Tumor characteristics (type, location, volume, percentage of supplying artery, presence of an early draining vein), angiographic extent of tumor devascularization, timing between embolization and surgery, estimated blood loss, and complication related embolization were evaluated.

Results: There were 37 tumors (mean volume, $90.9\pm83.6~\text{cm}^3$) composed of 18 meningiomas, six hemangioblastomas, six hemangiopericytomas, one metastasis, one osteoblastoma, one osteosacroma, one neurofibroma, one central neurocytoma, one glomus jugulare, one mixed oligoastrocytoma, and one glioblastoma multiforme. Early of draining veins were visualized in 24 patients (64.9%). Failure of pre-operative embolization occurred in four patients. Median time to surgery after embolization was seven days (ranged 1-171 days). There was statistically significant difference between grading of angiographic devascularization and estimated blood loss (p=0.009, Kruskal-Wallis test). Two patients (5.4%) had embolization-related complications, including hemorrhage during sub-selective catheterization and postoperative scalp necrosis.

Conclusion: Although pre-operative embolization of intra- and extracranial tumors was safe, only extensive or complete angiographic devascularization has been effective in less intra-operative blood loss. From this present study, indications regarding when to perform pre-operative embolization include history of excessive bleeding from previous surgery, known hypervascular tumor types (e.g., hemangiopericytoma, hemangioblastoma, paraganglioma), the presence of multiple flow voids on MRI, hypervascular tumors of skull or scalp, deep-seated tumors (e.g., cranial base tumor, intraventricular tumor) with difficulty in early surgical access of the main feeding vessels, and tumors associated with intratumoral aneurysm.

Keywords: Pre-operative embolization, Brain tumor, Cranial tumor, Meningioma, Hemangiopericytoma, Hemangioblastoma, Paraganglioma, Intra-axial tumor

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Pre-operative embolization of brain tumors, especially in meningioma, were performed to facilitate surgical resection by improving visualization, limiting intra-operative blood loss, which causes ischemic necrosis leading to softening of tumors and minimizing the pressure transmitted to the adjacent sensitive neural structures⁽¹⁻⁸⁾. The practice of embolization before surgery varies in different institutes. Although pre-operative embolization of

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meningiomas has been recommended as a standard procedure whenever possible^(9,10), it is not followed routinely due to the personal preferences of neurosurgeons⁽¹¹⁻¹⁴⁾. Considering high costs and the risk of complications following embolization, indiscriminate use of pre-operative embolization should be avoided^(15,16). The indications for pre-operative embolization of brain tumors, including meningioma and non-meningioma, are still unclear. Some studies demonstrated the results of pre-operative embolization in meningioma and other brain tumors^(11,17-19), whilst many studies evaluated only in meningioma^(1,2,4-6,10,12,13,15,16,20,21). The purpose of this present study is to evaluate the safety and efficacy of

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pre-operative embolization of intra- and extracranial tumors (i.e., meningiomas and non-meningiomas) and to determine the selection criteria of patients for presurgical devascularization.

Material and Method

Between June 2008 and August 2012, 37 patients (17 males, 20 females; mean age, 44.2±14.2 years), underwent pre-operative embolization for both intra- and extracranial tumors, were reviewed retrospectively in Prasat Neurological Institute (PNI). Computed tomography (CT) scan and magnetic resonance imaging (MRI) were obtained from all patients for the initial diagnosis of intra-and extracranial tumors. Tumor characteristics (type, location, volume, percentage of supplying artery, presence of early draining vein), angiographic extent of tumor devascularization, timing between embolization and surgery, estimated blood loss (EBL), and embolizationrelated complications were evaluated. All imaging studies were interpreted by neuroradiologists. The present study was approved by the ethics committee of the PNI.

Tumor volumes were measured in cubic cm (cm³) by analysis of the contrast-enhanced MRI using picture archiving and communication system (PACS; FUJIFILM, Stamford, CT, USA). The tumors were histologically typed according to the classification of tumors of the central nervous system based on the World Health Organization (WHO)(22). The anesthesia records were reviewed for estimated blood loss.

Diagnostic cerebral angiography and preoperative embolization were performed in the same session by the Seldinger technique via the femoral route under general anesthesia (GA) without any monitoring or provocative test. A biplane digital subtraction angiography system (AXIOM Artis FD Biplane Angiosuite; Siemens Medical Solutions, Erlangen, Germany) was used for all patients. Initially, the 5F Envoy catheter (Codman Neurovascular, Raynham, MA) was placed in the external carotid artery (ECA), the internal carotid artery (ICA), or the vertebral artery (VA) as a guiding catheter, then the microcatheter was coaxially advanced into the feeding vessel. Before insertion of the microcatheter, heparin at a dose of 100 IU/kg of body weight was administered intravenously when embolization via the ICA or the vertebrobasilar (VB) system, whilst embolization via ECA was performed without heparinization. The effects of heparin were reversed with protamine sulfate after completion of embolization. The tip of microcatheter

was navigated and positioned as close as possible to the tumor. Embolic material was injected through the microcatheter under real-time digital subtraction angiography (DSA).

The authors used two kinds of embolic materials, including n-butyl cyanoacrylate (NBCA; Histoacryl; B.Braun, Melsungen, Germany) and polyvinyl alcohol (PVA) particles (Contour, Boston Scientific, Natick, MA). Echelon microcatheter (eV3, Irvine, CA) or Prowler microcatheter (Codman, Raynham, MA) with NBCA and/or PVA particles (150-250 microns) was used for branches of ECA, whereas Magic microcatheter (Balt, Montmorency, France) with NBCA was used for branches of ICA or the VB system. Mixture of NBCA and an oil-based contrast agent (Lipiodol Ultra Fluid; Guerbet, Aulnay-sous-Bois, France) was prepared in proportions of 1:3 ratio of NBCA to Lipiodol. PVA particles were injected after dilution in contrast material (Hexabrix, Guerbet, Aulnaysous-Bois, France). Intravenous steroid was used in all patients for prevention of tumor swelling following embolization. After embolization, angiographic devascularization was divided into four grades: grade I-slight devascularization (<30%); grade II-moderate devascularization (30-60%); grade III-extensive devascularization (>60-90%); and grade IV-complete devascularization (>90%). Grading was determined by angiography of the ECA, the ICA, and/or the VA, which supplied the tumors.

Statistical analysis was performed with Statistical Package for the Social Sciences (SPSS) for Windows, version 16.0 (SPSS Inc., Chicago, IL, USA). Normally distributed data was presented as mean ± standard deviation (SD) and non-normally distributed data as median with interquartile range (p25-p75). Discrete variables were presented as frequencies and percentages. Comparison between grading of angiographic devascularization and estimated blood loss were analyzed using Kruskal-Wallis test. A probability value (*p*-value) less than 0.05 was considered statistically significant.

Results

Patient demographics, tumor characteristics, types of embolic agent, grading of angiographic devascularization, and volume of EBL are shown in Table 1. There were 37 tumors with mean volume, 90.9±83.6 cm³. Twelve patients (32.4%) were consulted for pre-operative embolization because of excessive blood loss prior surgery. Failure of embolization occurred in four patients (case 4, 12, 13, 29) due to

Table 1. Summary of demographic data, tumor characteristics, type of embolic materials, angiographic devascularization grading, and estimated blood loss

F/42 Yes F/35 Yes M/44 No F/63 No F/56 No F/56 No F/56 No F/50 Yes F/31 No F/30 No F/30 No M/60 No M/34 No M/34 No M/34 No M/39 No M/30 No M/3	Hemangiopericytoma Hemangiopericytoma Hemangiopericytoma Central neurocytoma Hemangiopericytoma Hemangiopericytoma Meningioma(transitional type) Glomusjugulare Atypical meningioma Meningioma(transitional type) Atypical meningioma Meningioma(transitional type) Meningioma(transitional type)	Sphenoid wing Occipital convexity						
F/27 Yes F/35 Yes M/44 No F/61 No F/56 No F/51 Yes F/41 No F/51 Yes F/41 No F/50 Yes F/40 Yes F/39 No M/60 No M/20 Yes F/54 No M/34 No M/34 No M/39 No M/47 No M/30 No M/47 No M/30 No M/47 No M/47 No M/47 No M/47 No M/47 No M/47 No M/47 No M/40 No M/47 No M/40 No M/47 No M/40 No M/47 No M/40 No M/55 No M/55 No	iopericytoma iopericytoma neurocytoma iopericytoma oma(transitional type) oma(transitional type) ugulare meningioma oma(transitional type) meningioma oma(microcystic type) meningioma	Occipital convexity	81.33	70/30/0	Yes	NBCA	2	5,000
F/35 Yes M/44 No F/63 No F/56 No F/51 Yes F/41 No F/51 Yes F/41 No F/50 Yes F/39 No F/50 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/34 No M/39 No M/39 No M/47 No M/30 No M/47 No M/30 No M/47 No M/47 No M/47 No M/47 No M/47 No M/47 No M/50 No M/47 No M/50	iopericytoma neurocytoma iopericytoma oma(transitional type) oma(transitional type) lgulare meningioma oma(transitional type) meningioma oma(microcystic type) meningioma	Comment of the commen	79.25	2/5/90	No	NBCA	4	006
M/44 No F/63 No F/56 No F/51 Yes F/41 No F/64 No F/31 No F/40 Yes F/39 No F/50 Yes F/50 Yes F/50 No M/60 No M/47 No M/39 No M/39 No M/39 No M/47 No M/30 No M/47 No M/47 No M/47 No M/47 Yes F/32 No M/47 No M/47 Yes F/32 No M/40 No M/30 No M/47 No M/40 No M/47 No M/40 No M/47 No M/40 No M/47 No M/40 No M/47 No M/40 No	neurocytoma iopericytoma oma(transitional type) oma(transitional type) lgulare meningioma oma(transitional type) meningioma oma(transitional type) oma(microcystic type)	Posterior fossa	32.00	5/0/95	Yes	NBCA/PVA	2	NS
F/41 No F/63 No F/56 No F/51 Yes F/41 No F/64 No F/31 No F/50 Yes F/39 No F/50 Yes F/50 No M/20 Yes F/54 No M/34 No M/39 No M/30 No M/30 No M/47 No M/30 No M/47 No M/47 No M/47 Yes F/32 No M/30 No M/30 No M/47 No M/40 No M/30 No M/47 No M/47 No M/47 No M/47 No M/47 No M/47 No M/47 No M/40 No M/47 No M/40 No M	oma(transitional type) oma(transitional type) ugulare meningioma oma(transitional type) meningioma oma(transitional type) oma(microcystic type) meningioma	Lateral ventricle	73.29	0/0/100	No			550
F/63 No F/56 No F/51 Yes F/41 No F/64 No F/31 No M/69 No F/30 No F/40 Yes F/54 No M/34 No M/34 No M/30 No M/30 No M/30 No M/47 Yes F/32 No M/30 No M/47 Yes F/32 No M/47 No M/47 Yes F/32 No M/30 No M	oma(transitional type) oma(transitional type) lgulare meningioma oma(transitional type) oma(microcystic type) meningioma	Falx	58.47	30/70/0	No	NBCA	1	2,000
F/56 No F/51 Yes F/41 No F/64 No F/31 No M/69 No F/30 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/30 No M/30 No M/30 No M/47 Yes F/32 No M/29 No M/47 Yes F/32 No M/30 No M/47 Yes F/32 No M/30 No M/30 No M/30 No M/47 Yes F/32 No M/30 No M/30 No M/30 No M/30 No M/30 No M/31 No M/30 N	oma(transitional type) gulare meningioma oma(transitional type) oma(microcystic type) meningioma	Velum interpositum	77.00	0/0/100	No	NBCA	3	1,850
F/51 Yes F/41 No F/64 No F/50 Yes F/39 No F/40 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/39 No M/30 No M/47 No M/30 No M/47 Yes F/32 No M/29 No M/47 Yes F/32 No M/29 No M/30 No M/30 No M/47 Yes F/32 No M/30 No M/30 No M/30 No M/30 No M/30 No M/30 No M/31 No M/30 No M/31 No M/32 No M/30 No M/30 No M/30 No M/31 No M/31 No M/32 No M/30 No M/31 No M/32 No M/30 No M/31 No M/32 No M/30 No M/31 No M/32 No M/32 No M/30 No M/31 No M/32 No M/30 N	neningiona oma(transitional type) oma(microcystic type)	Falx	44.55	20/80/0	Yes	NBCA	3	750
F/41 No F/64 No F/51 No M/69 No F/30 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/30 No M/30 No M/30 No M/47 Yes F/32 No M/29 No M/29 No M/30 No M/47 Yes F/32 No M/30 No M/31 No M/32 No M/30 No M/30 No M/30 No M/31 No M/30 No M	meningioma oma(transitional type) oma(microcystic type) meningioma	Femporal bone	82.99	80/10/10	Yes	NBCA/PVA	1	4,850
F/64 No F/31 No M/69 No F/30 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/34 No M/34 No M/39 No M/47 Yes F/32 No M/29 No M/29 No M/30 No M/47 Yes F/32 No M/47 Yes F/32 No M/29 No M/30 No M/30 No M/47 Yes M/30 No M/47 Yes M/30 No M/47 No M/30 No M/30 No M/30 No M/30 No M/30 No M/31 No M/30	oma(transitional type) oma(microcystic type) meningioma	Sphenoid wing	90.75	20/80/0	No	PVA	2	1,500
F/31 No M/69 No F/50 Yes F/39 No F/40 Yes M/60 No M/20 Yes F/58 No M/34 No M/34 No M/39 No M/30 No M/30 No M/47 Yes F/32 No M/29 No M/30 No M/30 No M/47 Yes M/47 Yes M/47 No M/29 No M/29 No M/30 No M/30 Yes M/30 No M/30 Yes M/30 No M/30 Yes M/31 No M/30 Yes M/31 No M/31 No	oma(microcystic type)	Sphenoid wing	182.78	60/40/0	Yes	NBCA/PVA	3	1,300
M/69 No F/50 Yes F/40 Yes M/60 No M/20 Yes F/54 No M/47 No M/30 No M/47 Yes F/32 No M/29 No M/29 No M/29 No M/29 No M/30 No M/47 Yes F/47 No M/29 No M/30 No M/30 No M/30 No M/30 No M/31 No M/30 No M/31 No M/32 No M/32 No M/30 No M/31 No M/31 No M/32 No M/31 No M/32 No M/32 No M/31 No M/32 No M/32 No M/31 No M/32 No M/31 No M/32 No M/32 No M/35 No M/35 No M/35 No M/35 No M/36 No M/37 No M/37 No M/37 No M/37 No M/38 No M/38 No M/31 No M/31 No M/32 No M/32 No M/34 No M/35 No M/36 No M/37 No M	meningioma	Sphenoid wing	123.96	70/30/0	Yes	PVA	3	2,600
F/50 Yes F/39 No F/40 Yes M/60 No M/20 Yes F/58 No M/34 No M/39 No M/30 No M/30 No M/47 Yes F/32 No M/29 No M/29 No M/29 No M/30 No M/30 No M/30 No M/30 No M/30 No M/31 No M/32 No M/32 No M/30 No M/31 No M/32 No M/32 No M/33 No M/34 No M/35 No M/35 No M/35 No M/35 No M/36 No M/37 No M/		Falx	33.00	20/20/0	Yes			800
F/39 No F/40 Yes M/60 No M/20 Yes F/58 No M/47 No F/54 No M/39 No M/47 Yes F/32 No M/29 No M/29 No M/29 No M/47 Yes F/47 No M/29 No M/29 No M/30 No M/30 No M/30 No M/30 No M/31 No M/31 No M/32 No M/32 No M/30 No M/31 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/31 No M/32 No M/31 No M/32 No M/31 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/32 No M/33 No M/34 No M/35 No M/36 No M/36 No M/37 No M/	ioblastoma	Posterior fossa	5.72	0/0/100	Yes			NS
F/40 Yes M/60 No M/20 Yes F/58 No M/34 No M/47 No F/54 No M/60 No M/60 No M/70 No M/29 No M/29 No M/29 No M/29 No M/29 No M/30 No M/30 No M/47 Yes F/47 No M/29 No M/30 No M/30 No M/30 No M/30 No M/31 No M/32 No M/30 No M/30 No M/31 No M/31 No M/32 No M/32 No M/30 No M/31 No M/32 No M/32 No M/31 No M/32 No M/31 No M/32 No M/31 No M/32 No M/32 No M/35 No M/36 No M/37 No	Meningioma(microcystic type)	Frontoparietal convexity	105.00	10/90/0	Yes	NBCA/PVA	1	1,500
M/60 No M/20 Yes F/58 No M/47 No F/54 No M/60 No M/60 No M/72 No M/29 No M/29 No M/29 No M/47 Yes F/47 No M/29 No M/55 No M/40 No M/55 No M/55 No	meningioma	Falx	243.05	10/90/0	No	NBCA	4	200
M/20 Yes F/58 No M/34 No M/47 No F/54 No M/60 No M/47 Yes F/32 No M/29 No M/29 No M/47 Yes F/47 No M/40 No M/55 No M/55 No M/55 No	ioblastoma	Posterior fossa	33.21	0/0/100	Yes	NBCA	3	006
F/58 No M/34 No M/47 No F/54 No M/60 No M/60 No M/47 Yes F/32 No M/29 No M/29 No M/29 No M/30 Yes F/47 No M/40 No M/55 No M/55 No	ioblastoma	Posterior fossa	72.00	10/0/90	Yes	NBCA	2	Ω
M/34 No M/47 No F/54 No M/39 No M/60 No M/47 Yes F/32 No M/29 No M/29 No M/47 Yes M/40 No M/55 No M/55 No M/55 No	Meningioma(fibrous type)	Falx	88.32	2/95/0	No	NBCA	4	009
M/47 No F/54 No M/39 No M/60 No M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/55 No	oma(metaplastic type)	Cribiform plate	3.51	0/100/0	Yes	NBCA	4	1,400
F/54 No M/39 No M/60 No M/30 No M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/55 No	iopericytoma	Posterior fossa	18.00	25/5/70	Yes	NBCA		3,050
M/39 No M/60 No M/30 No M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/55 No M/55 No	Meningioma(angiomatous type)	Sphenoid wing	50.92	40/60/0	Yes	NBCA	2	400
M/60 No M/30 No M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/55 No M/55 No	oma	Lateral ventricle	42.12	0/20/30	Yes	NBCA	4	450
M/30 No M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/55 No M/15 No	rcinoma	Skull	275.90	95/5/0	Yes	NBCA	4	450
M/47 Yes F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/28 Yes M/15 No		Skull	253.00	95/5/0	Yes	NBCA	4	2,650
F/32 No M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/28 Yes M/15 No	oma(transitional type)	Sphenoid wing	90.00	40/60/0	Yes	NBCA	2	3,400
M/29 No M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/28 Yes M/15 No	Meningioma(angiomatous type)	Falx	27.50	95/5/0	No	NBCA	3	800
M/30 Yes F/47 No M/67 Yes M/40 No M/55 No M/28 Yes M/15 No	Glioblastomamultiforme	Femporo-occipital	32.76	30/60/10	Yes	NBCA	_	1,500
F/47 No M/67 Yes M/40 No M/55 No M/28 Yes M/15 No		Scalp	144.00	100/0/0	Yes	NBCA	4	70
M/67 Yes M/40 No M/55 No M/28 Yes M/15 No	oma(transitional type)	Sphenoid wing	19.69	10/90/0	No			008
M/40 No M/55 No M/28 Yes M/15 No	ioblastoma	Posterior fossa	2.00	5/0/95	Yes	NBCA	4	200
M/55 No M/28 Yes M/15 No	ioblastoma	Posterior fossa	61.12	30/0/70	Yes	NBCA	2	2,350
M/28 Yes I M/15 No 0	Meningioma(microcystic type)	Frontal convexity	210.01	80/20/0	No	NBCA	2	4,000
M/15 No	oma(transitional type)	Sphenoid wing	131.40	60/40/0	Yes	NBCA/PVA	2	Ω
		Skull	350.65	90/10/0	No	NBCA	3	2,400
35 F/74 Yes Hemangioblastoma	ioblastoma	Posterior fossa	33.51	0/0/100	Yes	NBCA	3	150
F/44	ional type)	Sphenoid wing	123.75	90/10/0	No	PVA	4	009
37 M/34 No Meningioma(metapl	oma(metaplastic type)	Trigone	4.50	0/90/10	No	NBCA	4	150

ECA = external carotid artery; ICA = internal carotid artery; VB = vetebrobasilar system; EBL = estimated blood loss; NS = no surgery; U = unknown

inability to sub-select the target point of feeding vessels. Twenty-five patients underwent embolization with NBCA alone, three with PVA particles alone, and five with combination of NBCA and PVA particles. One patient (case 3) refused further surgery after embolization and lost to follow-up. Two patients (case 17, 33) were referred back to provincial hospitals for further surgery after embolization. One patient (case 13), who failed embolization, underwent radio surgery without surgery. Finally, remaining 33 patients were operated in the PNI. Median time to surgery after embolization was seven days (ranged 1-171 days). A follow-up MRI with Gadolinium after embolization was obtained in six patients for evaluation of necrotic areas. Pathological diagnosis revealed 18 meningiomas, six hemangioblastomas, five hemangiopericytoma, onemetastatic adenocarcinoma, one osteoblastoma, one osteosarcoma, one neurofibroma, one central neurocytoma, one glomus jugulare, one mixed oligoastrocytoma, and one glioblastoma multiforme (GBM). Histological diagnosis of meningiomas consisted the types of seven transitional, three atypical, three microcystic, two angiomatous, two metaplastic, and one fibrous. Nine tumors were located in the sphenoid wing, eight in the posterior fossa, six in the falx cerebri, three in the skull, three in the convexity, three in the ventricle (i.e., two lateral ventricles, and one trigone), and one each in the velum interpositum, the scalp, the temporal bone, the cribriform plate, and the temporo-occipital lobe. Therefore, there were 11 tumors located in the skull base (i.e., sphenoid wing, temporal bone, and cribriform plate).

The ratio of arterial supply to tumors revealed dominant supply from the ECA in 10 patients (27.03%), the ICA in 11 patients (29.73%), and the VB system in 11 patients (29.73%), whilst the mixed supply was from the ECA and ICA in five patients (13.51%). There were four patients (case 2, 8, 20, 27) that had tumors supplied from the ECA, ICA and VB system and two patients (case 22, 37) had tumors supplied from the ICA and VB system.

For eighteen meningiomas, a major supply was from the ECA in four patients (22.22%), the ICA in eight patients (44.44%), the VB system in one patient (5.56%), whilst the mixed supply was from the ECA and ICA in five patients (27.78%). In five out of six falcine meningiomas, dominant supply of tumors originated from the ICA.

For eleven skull base tumors (i.e., nine meningioma, one hemangiopericytoma, and one glomus jugulare), the major supply was from the ECA in five

patients, the ICA in three patients, whilst the mixed supply was from the ICA and ECA in three patients. Only one of three convexity tumors (i.e., one hemangiopericytoma, and two meningiomas) had dominant supply from the ECA (case 32).

All three calvarial tumors and one tumor of the scalp received blood supply mainly from the ECA. Intracranial tumors located in posterior fossa were fed dominantly from the VB system. Major arterial supply of intraventricular tumors was derived from choroidal branches of the ICA and/or VB system. All six hemangioblastomas had main arterial supply from the pial arteries of the VB system. Four tumors, that had pure supply from the VB system, included one central neurocytoma, one meningioma (velum interpositum), and two hemangioblastomas. Two intra-axial gliomas had dominant supply from the ICA.

Early of draining veins were visualized in 24 patients (64.9%). The presence of an early draining vein appeared in nine meningiomas (i.e., four transitional, two microcystic, one angiomatous, one atypical, and one metaplastic type), six hemangioblastomas, three hemangiopericytomas, two calvarial tumors (i.e., one metastasis, and one osteoblastoma), one GBM, one mixed oligoastrocytoma, one neurofibroma, and one glomus jugulare.

There was statistically significant difference between grading of angiographic devascularization and EBL (p=0.009, Kruskal-Wallis test). Median volume of EBL was 2,000 ml (ranged 1,500-3,950 ml) in grade I; 2,875 ml (ranged 1,225-4,250 ml) in grade II; 1,100 ml (ranged 762-2,262 ml) in grade III; and 500 ml (ranged 200-900 ml) in grade IV.

Embolization-related complications occurred in two patients (5.4%). One patient (case 18) had extravasation of contrast material during super selective angiography of feeding vessel. This defect was sealed immediately with NBCA and the patient had no neurological deficit occurred. Another patient (case 32) developed post-operative necrotic scars after embolization via superficial temporal artery. However, this problem was solved with only wound dressing.

There were two surgery-related complications. Post-operative bleeding occurred in one patient (case 1) then undergoing removal of blood clot. Another complication was post-operative wound infection in one patient (case 8) who received surgical drainage and antibiotic therapy. There was no mortality in this present study.

Either early post operative or late post operative MRI was obtained for evaluation of residual

tumor in all 33 remaining patients operated in the PNI. Tumor removal was total in 12 patients, near total in nine patients, and sub-total in 12 patients. Conventional radiotherapy was performed in eight tumors, including one metastatic adenocarcinoma, one GBM, four hemangiopericytomas, one osteosarcoma, and one mixed oligoastrocytoma. Twelve tumors (i.e., nine meningiomas, one glomus tumor, one hemangioblastoma, and one central neurocytoma) were treated with stereotactic radio surgery.

Discussion

Goals of pre-operative embolization of brain tumors are reduction of tumor vascularity and control of inaccessible arterial supply. To avoid inadvertent embolization of normal brain parenchyma and cranial nerves, meticulous evaluation of vascular anatomy or angio-architecture of cranial tumors should be carried out by selective angiography^(3,14,21,23). During the past several decades, interventional neuroradiology has been evolved in the field of technologies, e.g., flowdependent microcatheter, high resolution digital subtraction angiography with road-mapping capability, and new embolic materials^(24,25). The use of appropriate embolic materials is important in maximizing the effectiveness of presurgical embolization and minimizing the risk the complications related to embolization(26).

Embolic materials used for pre-operative embolization of brain tumors are mainly categorized into three groups, i.e., particulate embolic materials, liquid embolic agents, and mechanical devices. Particles are the most commonly used embolic material, including PVA particles, gelatin sponge (gelfoam), and gelatin microspheres^(6,12,14,16,20,27). PVA particles and gelatin microspheres are non-absorbable, and they likely produce permanent occlusion. Gelfoam is reabsorbable, then the embolized vessels can recanalize within a few days⁽⁵⁾. Particulate materials are radiolucent and require a mixture of contrast agent to monitor embolization under fluoroscopic guidance by slowly injection until the stasis of antegrade flow was achieved. Wakhloo et al⁽²⁷⁾ evaluated the efficacy of pre-operative meningioma devascularization mainly via external carotid artery, by comparison between large PVA particles (150 to 300 microns) and small PVA particles (50 to 150 microns). The study found that small particles are more likely to reach in distal precapillary tumor-bed leading to higher rate of tumor necrosis confirmed by post-contrast MRI and histopathological findings, whereas large particles are more likely to lodge proximally in the large

feeding vessels resulting in revascularization of the tumor by collateral meningeal vessels bypassing the site of occlusion. Besides, the irregular surface of large PVA particles has a high coefficient of friction permitting the particles to rest against the vessel wall with incomplete occlusion causing stagnation of the blood flow and producing a combination of PVA and blood clot that will eventually recanalize with reendothelialization of the non-absorbent PVA after clot lysis. However, in the large-scale study by Carli et al⁽¹³⁾ found that the use of small PVA particles (45 to 150 microns) proved to be the only significant risk factor for hemorrhagic complications, and discontinued using small particles for routine embolization. Another comparative clinical study, between gelatin microspheres (100 to 300 microns), PVA particles (45 to 150 microns), and PVA particles (150 to 250 microns) by Bendszus et al⁽²⁰⁾, showed that gelatin microspheres significantly penetrated the vascular bed of tumor deeper than PVA particles of either size, in which produce significantly less intra-operative blood loss. This due to the different biomechanical properties, including surface structure, deformability, and regularity of shape. Nevertheless, PVA particles are still the most commonly used agents among particulate embolic materials. Liquid embolic agents can penetrate deeper into the vascular bed of tumors than particles, leading to create more tumor necrosis(28). Practically, liquid embolic agents for pre-surgical embolization consist of NBCA and Onyx (eV3, Plymouth, MN, USA). NBCA is a fast polymerizing agent, which polymerizes rapidly after contact with ionic solutions (e.g., blood), and suitable for high flow fistula in brain arteriovenous malformations⁽²⁹⁾. Few studies recommended using NBCA as the first-choice embolic material for preoperative embolization of tumors(11,30). When the microcatheter cannot be navigated close enough to the tumor or beyond potential anastomoses, the use of NBCA should be avoided⁽³⁰⁾. Moreover, embolization with NBCA requires significant expertise for precise delivery of this agent⁽³¹⁾. Gore et al⁽¹⁷⁾ reported the off-label use of Onyx, which is a bio-compatible liquid polymer composing of the ethylene-vinyl alcohol copolymer (EVOH) suspended in dimethyl-sulfoxide (DMSO) and mixed with tantalum powder, using for pre-operative embolization of cranial and spinal tumors and showed that the results were excellent with no embolization-related complications. Both NBCA and Onyx are permanently occlusive agents and have radiopacity for easy visualization during embolization. Opposite to NBCA, Onyx has non-adhesive property making Onyx a more controllable agent than NBCA⁽³²⁾. Even though Onyx needs fewer arterial catheterizations, it tends to require prolonged fluoroscopic times resulting in more radiation exposure than NBCA. Mechanical devices (e.g., coil and balloon) were used as adjunct materials to occlude large proximal feeders with dangerous anastomoses or to occlude the ICA or the VA temporarily to change the flow during injection of embolic materials depends on the duration of occlusion desired and individual physician preferences. Ideally, embolic materials should penetrate distally into the tumor capillary beds with promoting tumor necrosis whilst preserving the normal surrounding tissues^(2,7,34).

A preliminary report by Horton et al⁽³⁵⁾ showed that a provocative test by injection lidocaine into external carotid branches to test cranial nerve function is important. This test can increase the safety of therapeutic embolization with small particles or liquid agent. There was reflux of lidocaine into the posterior circulation causing temporary alteration in neurologic function in one patient. Another protocol of provocative test in the external carotid territory was developed for avoiding complications from injection of lidocaine into the cerebral circulation⁽³⁶⁾. The provocative test with amobarbital sodium was used to reveal dangerous anastomoses from external carotid branches to the cerebral circulation, and testing with lidocaine was followed to show vascular supply to cranial nerves. When positive lidocaine test was encountered, embolization was performed by using large particles or coils(35,36). However, even though the result of provocative test is negative, the chance of reflux of embolic material into unwanted territories may occur, in which prevention of this situation requires extensive experience of neurointerventionist and knowledge of the related anatomy. Other concerns about using provocative test were adding time to procedure and false-negative results(35,37). Gupta et al(7) reported two patients who developed blindness after embolization with PVA particles despite passing provocative testing. Many studies^(3,8,10,20,27,30,38-41) preferred embolization under local anesthesia or neuroleptic anesthesia with or without the provocative test. One study used provocative test only in selected tumors adjacent to the motor cortex⁽¹⁹⁾. Embolization was also performed under GA for high quality selective angiograms to obtain the best possible anatomic analysis during the most critical moments of embolization, and did not use provocative testing in several studies(23,26,32,42,43). Some studies used somatosensory evoked monitoring and electroencephalography under GA(11,17). Pre-operative embolization in large hypervascular brain tumors supplied by pial branches may be preferred to perform under GA⁽¹⁸⁾. Intratumoral injection by direct percutaneous puncture technique should be performed under GA because this procedure is extremely painful(44,45). In pediatric patients, who are uncooperative, pre-operative embolization also should be done under $GA^{(14)}$. Fortunately, there were no complications of stroke or cranial nerve palsies in this present study. For injection with PVA particles, the authors chose particles ranging from 150 to 250 microns in size because using particles larger than 150 microns is safe to avoid penetration to either potentially dangerous vessels (i.e., dangerous anastomoses between ECA and ICA or VA) or arteries supplying to cranial nerves (i.e., vasa nervorum)(3,21,46). Whilst injection with NBCA, catheterization of tumor feeders has to be performed as close as possible to the tumor or distal to the origin of the dangerous vessels⁽³⁰⁾. Super selective catheterization is a major role to avoid inadvertent embolization of dangerous anastomoses. The study of pre-operative embolization of paragangliomas under GA by Valavanis(23) reported arterial anastomoses of the ascending pharyngeal artery (APhA) or the occipital artery (OA) with the VA in 34% of all cases. In the present study, anastomoses between extra-and intracranial arteries were angiographically identified before embolization in five (13.5%) of 37 patients (Fig. 1, 2H, 3F).

Intra- and extracranial tumors in this present study have mono- or multicompartmental vascular composition, which the majority of these tumors (92%) have multiple compartmental supply. Multicompartmental tumors, especially in large size, are supplied by different vessels for individual compartments (Fig. 4D, 4H, 4I, 5C, 5D, 6E, 6F). Monocompartmental tumors may be completely devascularized by embolization through one feeding artery (Fig. 7C, 8D). To achieve effective devascularization of multicompartmental tumors, each compartment should be embolized through all individual feeders (Fig. 6G, 6H, 9G, 9H).

Based on the location of the tumor, arteries supplying meningiomas usually derived from dural arteries and/or pial arteries. Dural arterial supply consists of branches from the ECA (i.e., APhA, OA, middle meningeal artery (MMA), accessory meningeal artery), ICA (i.e., inferolateral trunk (ILT), meningohypophyseal trunk (MHT), ethmoidal branches of the ophthalmic artery (OphA)), or VB system (i.e.,

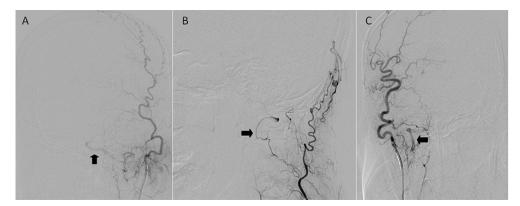


Fig. 1 (A) AP view of the left OA injection in falcine meningioma (case 26). (B) Lateral view of the right deep cervical artery injection in neurofibroma of the scalp (case 28). (C) AP view of the right OA injection in hemangiopericytoma of the posterior fossa (case 20). Anastomoses between muscular branches of occipital and deep cervical arteries and the ipsilateral VA (arrows) at C2 level were illustrated in these three cases.

posterior meningeal artery)(3,7,9,21,26). The pial supply is provided by cortical branches from the ICA (i.e., anterior cerebral artery (ACA), middle cerebral artery (MCA)), or VB (i.e., posterior cerebral artery (PCA), superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA))(9,18,19,26,47). Moreover, the superficial temporal artery (STA) may contribute to the tumor, either by directly supplying the subgaleal portion or by transosseous supplying to the intradural portion (Fig. 10E, 10F)⁽²¹⁾. The proportions of arterial supply in meningiomas varied in published studies. Some previous studies(10,13,20), which the majority of meningiomas were located in convexity of dura, falx, or tentorium, showed that most patients had meningiomas supplied dominantly from the ECA. Nevertheless, other studies^(6,34) of meningiomas in similar locations found that there were meningiomas supplied dominantly by the ECA in less than 50% of the patients. On the other hand, a series of only skull base meningiomas⁽²¹⁾ revealed that blood supply originated purely from the ICA or from combination of the ECA, ICA, and VA in the majority of the patients. Because meningiomas in this present study were located commonly in the skull base. Then, the results of the present study showed that the ratio of arterial supply of most meningiomas was a major supply from the ICA or mixed supply from the ECA and ICA. The pattern of tumor supplying in other tumors (non-meningiomas) also derives from these three main systems (i.e., the ECA, ICA, and VB system) depended on the location of the tumors. For example, posterior fossa tumors were supplied by the VB system and/or ECA (Fig. 6B), calvarial tumors mainly by the ECA (Fig. 9D, 9E), and intraventricular tumor by

the choroidal branches of the VB system and/or ICA.

At the beginning, many studies suggested to perform pre-operative embolization of meningiomas exclusively supplied by external carotid branches without embolization via branches of the ICA, especially pial branches because of the risk of stroke^(1-3,5-7,9,10,12,13,20,26,34,38,48). For example, in this present study, the patient (case 36) with large hypervascular sphenoid wing meningioma was embolized only via the ECA with PVA particles (Fig. 11D). Meningiomas with mixed supplying from external and internal carotid arteries have been reported to show increased flow from residual feeders of the ICA after embolization of the external carotid branches, then embolization of this situation may be of limited benefit or even contraindicated^(3,34,49). The report of Teasdale et al⁽³⁴⁾ demonstrated increasing of the blood flow and volume found in radioisotope studies, and showed that profuse hemorrhage occurred only in tumors having significant internal carotid feeding vessels after embolization of external carotid feeders. There was a report of a patient who died from uncontrollable bleeding during surgery after embolization via the ECA in a convexity meningioma with equal external and internal carotid supply⁽⁴⁾. Later, more studies were published in preoperative embolization via branches of the ICA, including OphA, anterior choroidal artery (AChA), MHT, ILT, and pial vessels(11,14,18,19,21,37,47,50-52). Due to higher rate of particulate embolization-related hemorrhage, the use of NBCA for devascularization of the intra-axial tumor vessels in branches from the ICA or VB system was recommended(14). In the present study, the authors prefer to use NBCA for internal carotid

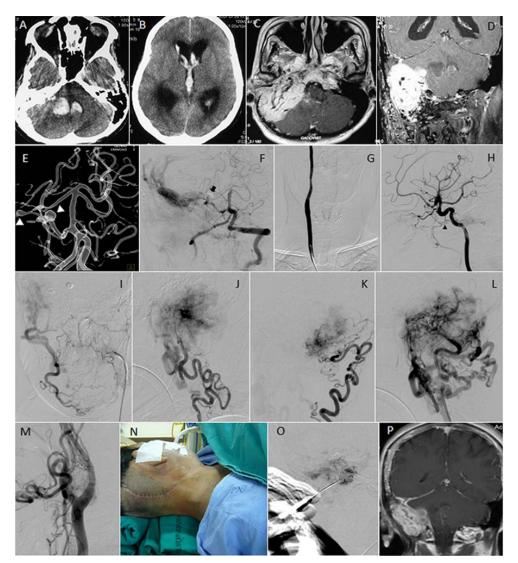


Fig. 2 Glomus jugulare tumor (case 8): (A&B), CT scan showed hemorrhage at right middle cerebellar peduncle with IVH. Axial (C) and coronal (D) T1-weighted Gd-enhanced MRI showed a large homogeneously enhancing tumor with multiple flow voids within the tumor locating extradurally in right temporal bone, mastoid, and posterior petrous bone with extension intradurally into posterior fossa. (E), 3D reconstruction of left vertebral angiography, AP view, revealed two right AICA aneurysms (white arrowheads). The proximal and larger one is probably the rupture site. (F), AP view of left VA injection illustrated prominent tumor blush fed by the right AICA with early venous draining to the right sigmoid sinus. Of note, coil pack (black arrow) is within the proximal right AICA aneurysm. The tumor and distal AICA aneurysm were simultaneously embolized via the right AICA (not shown). (G), Right CCA injection, AP view, revealed only right ICA because of prior ligation of right ECA from other hospital ten years ago. (H), Lateral view of the right ICA injection showed reconstitution of the right IMA from an artery of the foramen rotundum (black arrowhead). (I), Left ECA injection, APview, demonstrated reconstitution of the right lingual, facial, ascending palatine, and internal maxillary arteries from the contralateral counterparts. Lateral views of right ascending cervical artery (J), right deep cervical artery (K), and right VA (L) injection demonstrated prominent tumor stain supplied by the reconstituted right APhA, PAA, and OA. (M), Lateral view of left CCA injection showed splaying of the bifurcation with tumor blush, representing a carotid body tumor. (N), the patient's position before percutaneous tumor puncture. (O), Intratumoral injection showed tumor parenchymography. After seven intratumoral injections with NBCA were performed, the patient then underwent radiosurgery for the residual tumor. (P), Coronal T1-weighted Gd-enhanced MRI showed a residual tumor two years after radiosurgery.

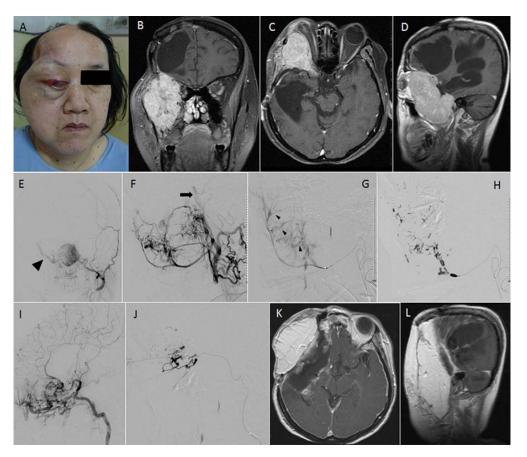


Fig. 3 Recurrent hemangiopericytoma (case 1): (A), A photograph of the patient illustrated right facial swelling, right proptosis with neurotrophic keratitis, and radiation induced alopecia. Coronal (B), axial (C), and sagittal (D) T1weighted Gd-enhanced MRI showed large homogeneously enhancing mass involving right infratemporal fossa with intraorbital invasion causing significant compression of right orbital contents. Cystic encephalomalacic changes were noted in the right frontal, temporal, and parietal lobes. (E), AP view of left ECA injection revealed prominent tumor stain fed by left IMA. Early draining vein (arrowhead) was also noted. (F), Right ascending cervical injection, lateral view, showed tumor blushes supplied by reconstituted hypertrophic right transverse facial artery, right IMA, and right APhA due to ligation of the right external carotid artery from prior surgery. Of note, VB system (black arrow) is from anastomosis between muscular branches of the right ascending cervical artery and ipsilateral VA. (G), Superselective right transverse facial artery injection demonstrated large compartment of hypervascular tumor blush in right infratemporal fossa. Corkscrew vessels (small arrowheads) were noted. (H), The NBCA injection via right transverse facial artery showed distal distribution of NBCA. (I), lateral view of left ICA injection revealed superior and intraorbital compartment of tumor supplied mainly by the right OphA. (J), The tumor was embolized with NBCA via the right OphA. After reconstructive surgery with enucleation, postoperative axial (K) and sagittal (L) T1-weighted Gd-enchanced MRI showed near total resection of the tumor and myocutaneous flap.

and vertebrobasilar branches. For external carotid branches, the authors can use either PVA particles or NBCA.

The OphA is a functionally important vessel, which requires knowing the appropriate knowledge of the anatomy. The intraorbital course of the OphA is divided into three portions by the central retinal artery originates from the distal part of the first portion of the

OphA⁽⁵³⁾. Super selective embolization via the OphA should catheterize distally beyond the second portion of the intraorbital OphA (i.e., the bend around the optic nerve) and gentle injection of embolic materials without reflux are mandatory to avoid damaging to visual pathways⁽⁴²⁾. There was a report of visual complications from using gelfoam powder through the OphA that may be caused by releasing of a small

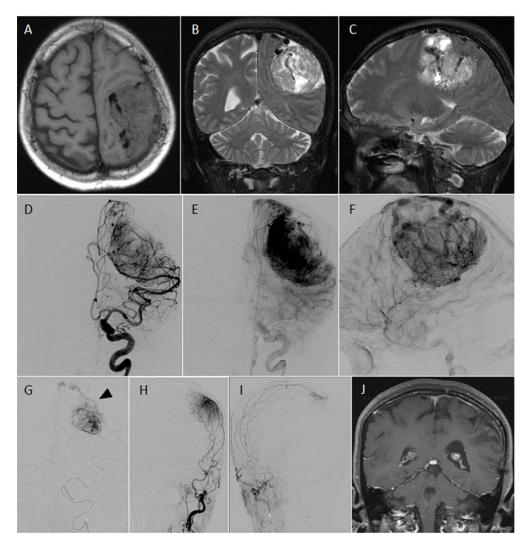


Fig. 4 Convexity meningioma (case 14): Axial T1-weighted (A), coronal T2-weighted (B), and Sagittal T2-weighted (C) MRI showed large left frontoparietal tumor with multiple flow voids in and around the tumor. (D), AP view of left ICA injection demonstrated tumor blush fed mainly by pial branches of left MCA and left ACA. AP (E) and lateral (F) views, capillary phase, revealed high tumor vascularity and large draining vein from the tumor into the superior sagittal sinus. (G), Superselective angiography of the pial branch of the left MCA illustrated inferomedial compartment of tumor stain with early draining vein (arrowhead). AP views of the left MMA (H) and right MMA (I) injections showed small superior compartment of tumor stain supplied by ipsilateral and contralateral MMAs. (J), Post-operative T1-weighted Gd-enhanced coronal MRI confirmed total removal of the tumor.

residual amount of gelfoam powder within microcatheter into the central retinal artery during super selective angiography after embolization⁽⁵⁴⁾. Alvarez et al⁽⁵⁵⁾ reported successful embolization through the OphA using NBCA without any complications and suggested that the quality of the imaging to provide the details of the safety point (i.e., the point beyond the second part of the OphA) and the ideal point (i.e., the point nearest the lesion) was mandatory before beginning

the treatment. Later, Matsumaru et al⁽⁴²⁾ also reported successful embolization of the OphA using NBCA in larger series from the same center under GA without functional testing. They discouraged the use of particles in this territory and suggested that NBCA was the best embolic material due to its low viscosity. Trivelatto et al⁽³²⁾ used Onyx for pre-operative embolization of meningiomas fed by the OphA without the provocative test, and showed that no patient experienced decreased

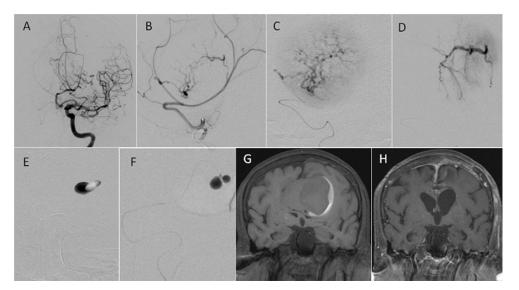


Fig. 5 Falcine meningioma (case 18): AP (A) and lateral (B) views of the left ICA injection showed a tumor blush supplied mainly by branches of the left pericalloal artery. Lateral views of superselective angiography of anterior branch (C) and posterior branch (D) of the left pericallosal artery demonstrated anterior and posterior compartment of tumor stain, respectively. (E), Superselective left pericallosal branch injection, AP view, revealed extravasated contrast material. (F), The NBCA was immediately injected to stop contrast extravasation. Pre-operative embolization of this patient was achieved one week later. (G), One day after embolization, T1-weighted coronal MRI disclosed left peritumoral hypersignal intensity representing subacute hemorrhage. (H), Post-operative T1-weighted Gd-enhanced coronal MRI confirmed total removal of the tumor.

visual function after embolization but peritumoral hemorrhage occurred in one of the five patients. In the present study, the authors preferred to use NBCA in embolization via the OphA after navigating of the tip of a microcatheter to the proper position (Fig. 3J, 8C). However, in one patient (case 29), the tip of a microcatheter could not be advanced to the appropriate position, embolization then was terminated due to risk of visual complications (Fig. 12D).

The first report of pre-operative embolization through the AChA by Oyama et al(52) showed successful embolization with particles in large trigonal meningioma. The AChA consists of cisternal and plexal segments. Successful embolization via this artery requires the microcatheter to enter the plexal segment beyond the plexal point (i.e., the point of entry into the choroid plexus). However, embolization through the AChA should be performed with caution because it can cause major neurological deficits. There was a report of hemiparesis and homonymous hemianopia developed after embolization via the AChA with PVA particles and gelfoam powder and the authors suggested that it was difficult to inject PVA particles without reflux⁽⁵⁴⁾. Pre-surgical embolization of trigonal meningioma (case 37) via the AChA with NBCA was

achieved in this present study (Fig. 13D).

Kaji et al⁽⁴⁷⁾ initially reported successful preoperative embolizations of cortical branches of the ICAs in meningiomas with particles. The study proposed four criteria for embolization of pial branches, including purely or predominantly supply from the ICA, no staining of the adjacent normal brain territories in super selective angiography, avoiding the dominant hemisphere for speech by using the amobarbital test, and advancing of microcatheter just proximal to the tumor. Later, Hirohata et al⁽¹⁸⁾ also reported successful preoperative embolizations of large hypervascular brain tumors via pial branches of the ICAs under GA without provocative testing. In the present study, pial branches of the ICA or VB system were successfully embolized by using NBCA in several cases (Fig. 6I, 14C).

The optimal timing between tumor embolization and surgery has been debated. Subsequent surgery was performed one to seven days after embolization to avoid the risk of recanalization of the feeding vessels^(2,6,13,14,16,20,27,34). There are two comparative researches studying about the appropriate surgical timing after embolization. First, Chun et al⁽⁴⁸⁾ reviewed in 50 patients who underwent pre-operative embolization only via branches of the ECA with mainly

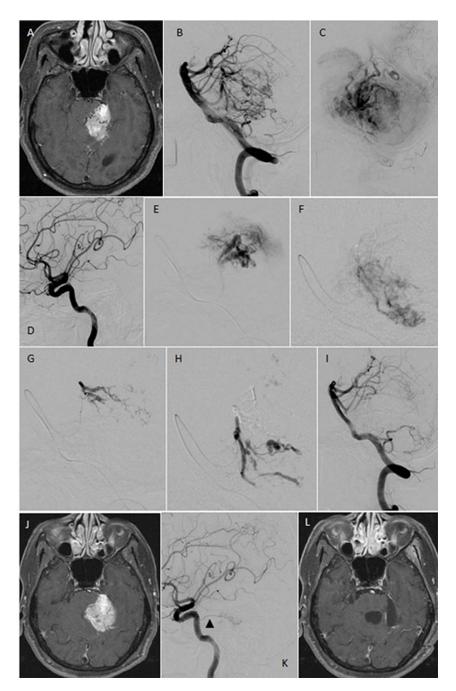


Fig. 6 CPA hemangioblastoma (case 16): (A), T1-weighted Gd-enhanced axial MRI showed heterogeneously enhancing tumor with multiple flow voids at left CPA. Arterial (B) and venous (C) phase of left VA, lateral view, demonstrated prominent tumor stain and dilated draining veins. D, There was no feeder vessel identified from left ICA. Superselective left SCA injection showed tumor blush of upper (E) and lower (F) compartment. Glue cast deeply penetrated feeding pedicles in upper (G) and lower (H) compartment. (I), Post-embolization angiography revealed nearly complete angiographic devascularization. The patient felt better after embolization and refused surgery. (J), Two months after embolization, MRI indicated tumor progression, even though flow voids seem to be decreased. (K), A follow-up left ICA injection, lateral view, illustrated the new tumor feeder (arrowhead) recruited from left MHT. This patient was operated on 171 days after embolization with an EBL less than 1,000 ml. (L), Post-operative T1-weighted Gd-enhanced axial MRI confirmed complete resection of the tumor.

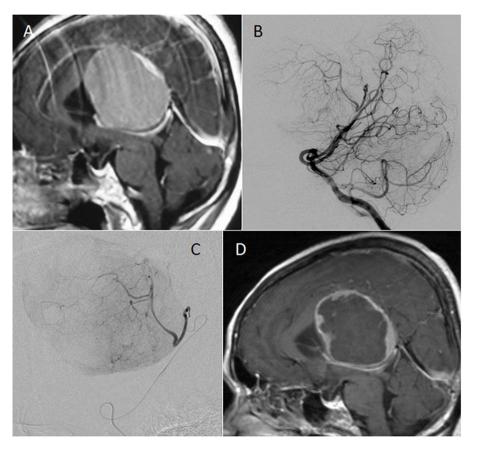


Fig. 7. Meningioma of the velum interpositum (case 6): (A), T1-weighted Gd-enhanced sagittal MRI before embolization showed large homogeneously enhancing tumor. (B), Left VA injection, lateral view, revealed tumor blush supplied mainly by pial branch arising from left PCA. (C), Superselective angiography of the pial branch of left PCA before embolization, late arterial phase, illustrated nearly entire tumor staining. (D), One week after embolization, T1-weighted Gd-enhanced sagittal MRI demonstrated marked reduction in enhancing of the tumor.

using PVA particles, and demonstrated that delayed resection of meningiomas more than 24 hours after embolization resulted in less intraoperative blood loss without clearly addressing the optimal duration of the delay. Another research by Kai et al(38) studied in 42 patients with meningiomas received more than 50% of their blood supply from the ECA embolized with nonabsorbable particles. The study concluded that the optimal interval was seven to nine days because this interval allowed the greatest degree of tumor softening. Shi et al⁽²⁸⁾ waited for ten days after emboliztion of three meningiomas with Onyx, and suggested that it was safe to delay surgical resection after embolization with Onyx. Wang et al(14) evaluated the outcomes of pre-operative devascularization of pediatric intra-axial and intraventricular hypervascular brain tumors. The study suggested performing the embolization and surgery in a single session to reduce the risk of delayed

tumor bleeding after embolization. Eskridge et al⁽⁸⁾ reported the patient with a large posterior fossa hemangioblastoma and hydrocephalus worsened clinically six hours after embolization requiring emergency ventriculostomy and tumor removal. They believed that the cause was probably from worsening of obstructive hydrocephalus resulting from tumor swelling. Peritumoral brain edema was significantly extensive when the tumor received a vascular supply from the pial arteries⁽⁵⁶⁾. An increase in volume of the tumor after embolization can occur and produce neurological deficits, which the use of steroids before and after embolization is suggested to prevent this effect^(8,27). To avoid the risk of brain edema with herniation caused by tumor swelling after embolization, resection of large hypervascular brain tumors fed by the pial vessels should be performed immediately after embolization⁽¹⁸⁾. In the present study, timing to

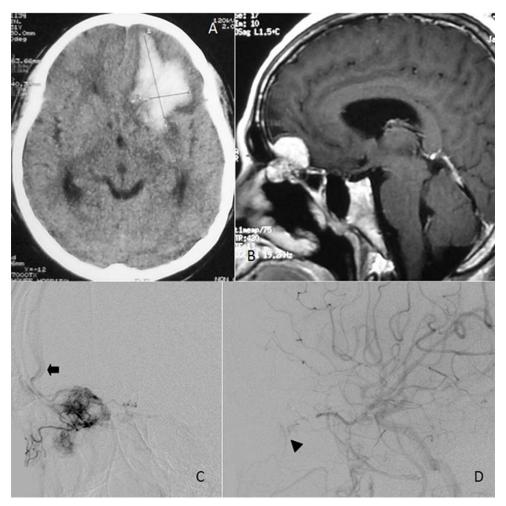


Fig. 8 Cribriform plate meningioma (case 19): (A), CT scan revealed left frontal hematoma. (B), Sagittal T1-weighted Gdenhanced MRI showed homogeneously enhancing tumor involving the cribriform plate and ethmoid sinus. (C), Lateral view of selective left OphA injection demonstrated tumor blush supplied by anterior ethmoidal branch. An early draining (black arrow) to the superior sagittal sinus was noted. (D), Post-embolization angiography of the left ICA, lateral view, confirmed grade IV angiographic devascularization with preservation of the choroidal crescent (arrowhead).

surgery after embolization depends on embolic material chosen. NBCA is more permanent than PVA particles, and may last for weeks or months; whilst particles can degrade over time, increasing the possibility of recanalization of occluded feeding vessels⁽¹¹⁾. If surgery is planned within a few days after embolization, selection of particles is reasonable⁽⁷⁾. After embolization with PVA particles, subsequent surgery should not be delayed for more than two weeks⁽⁵⁷⁾. For this present study, the patients with large supratentorial tumors and midline shift, surgical procedures were performed on the first day after embolization (Fig. 11B, 14A).

Koike et al⁽⁵⁸⁾ reported patient with parasagittal

meningioma exclusively supplied by bilateral MMAs embolized completely with non-absorbable particles resulting in significant reduction in tumor size and long-term remission of clinical symptoms without subsequent surgery. However, a follow-up imaging of this patient indicated tumor regrowth four years after embolization. Wakhloo et al⁽²⁷⁾ also reported complete recovery from neurological deficits in patient with convexity meningioma shrinking after therapeutic embolization with small particles without surgery. Another similar patient demonstrated by Probst et al⁽²⁾. The results found that the necrotic areas of the clival meningioma embolized with fibrin glue were resorbed, and then

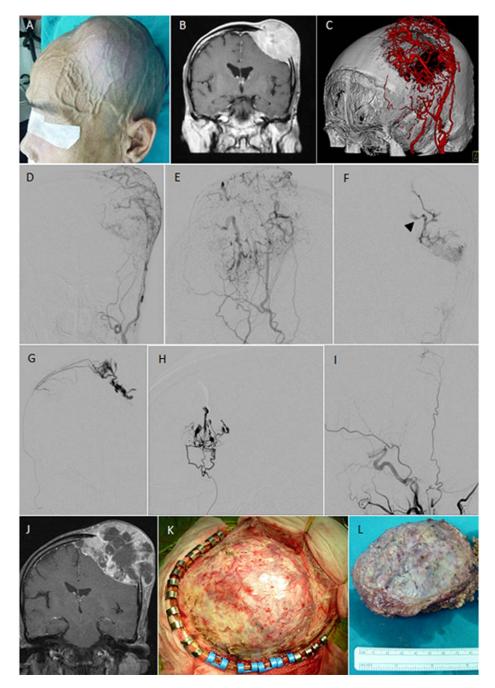


Fig. 9 Skull metastasis (case 23): (A), The patient presented with large skull mass at left frontoparietal area. (B), Coronal T1-weighted Gd-enhanced MRI showed large homogeneously enhancing mass at left parietal bone. (C), 3D reconstructive angiography illustrated skull defect and feeding vessels of hypervascular tumor (red color). AP (D) and lateral (E) views of left MMA injection showed strong tumor blush supplied mainly by hypertrophicbranches of left STA and MMA. (F), AP view of superselective angiogram of left MMA demonstrated tumor blush and early venous drainage (arrowhead). AP (G) and lateral views (H) during glue injection at right and left MMAs, respectively. (I), Lateral view of right CCA confirmed grade IV angiographic devascularization. (J), Coronal T1-weighted Gd-enhanced MRI after the embolization demonstrated marked reduction in enhancing of the tumor. (K), Intraoperative view revealed minimal blood loss during surgery. (L), A photograph showed macroscopic specimen of the tumor after en-bloc resection.

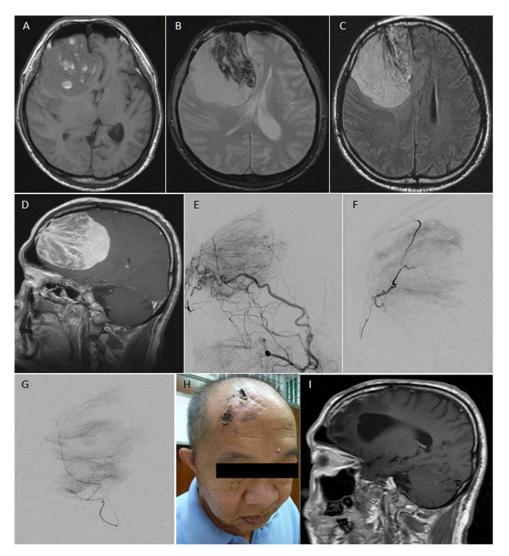


Fig. 10 Convexity meningioma (case 32): (A), Preoperative T1-weighted axial MRI showed a large right frontal tumor composed of mixed hypersignal and isosignal intensities. (B), T2-weighted gradient-echo axial MRI revealed hyposignal intensity representing hemorrhage in anteromedial part of the tumor. (C), FLAIR-weighted axial MRI demonstrated intratumoral linear flow voids radiating outwards from the central vascular pedicle at the right frontal skull. (D), T1-weighted Gd-enhanced sagittal MRI showed heterogeneously enhancing tumor. (E), Lateral view of right ECA injection revealed sunburst centrifugal tumor blush supplied mainly by right STA and MMA. Superselective angiography of right STA (F) and right MMA (G) demonstrated prominent tumor stains in late arterial phase. (H), One month post-operative picture of the patient showed dry necrotic scars, which healed itself later without surgical intervention. (I), Post-operative T1-weighted Gd-enhanced sagittal MRI confirmed nearly complete tumor removal.

markedly decreased in tumor size. This meningioma was removed in six months later. By using MR spectroscopy instead of pathological verification, Bendszus et al⁽⁴⁰⁾ studied prospectively in seven patients with meningiomas embolized alone without surgery, and reported substantial shrinkage of tumor occurred in six patients except one tumor unchanged

despite complete angiographic devascularization, in which was speculated that the tumor vessels recanalized because of proximal occlusion. Subsequently, Bateman et al⁽⁵⁹⁾ proposed the concept of definitive embolization of meningiomas (i.e., treating them by embolization without subsequent surgery) after concerning about complications of surgery and radio

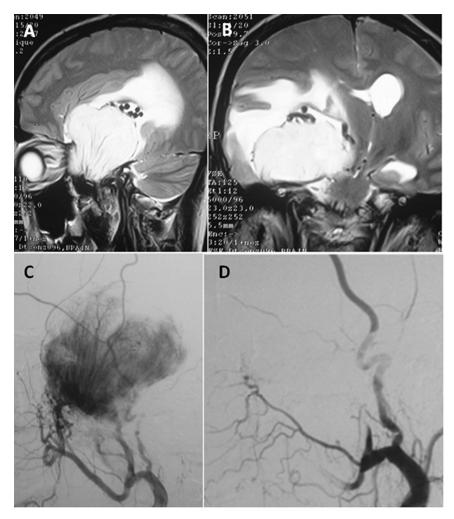


Fig. 11 Sphenoid wing meningioma (case 36): Sagittal (A) and Coronal (B) T2-weighted MRI showed multiple signal voids in and around the large right sphenoid wing tumor with peritumoral edema and marked midline shift. (C), Lateral view of right ECA injection demonstrated a prominent tumor stain supplied mainly by distal IMA and MMA. (D), Right external carotid artery injection after pre-operative embolization with PVA particles revealed complete angiographic devascularization of the tumor with preservation of the STA.

surgery. For non-meningiomas, Casasco et al⁽⁴⁴⁾ presented palliative embolization for three metastases of the clavarium by a direct percutaneous puncture technique with alcohol resulting in good tumor remission. Another glomus tumor was embolized with NBCA by intratumoral injection led to reduction in size and being stable after eight months of follow-up. Dabus et al⁽³¹⁾ also reported a palliative embolization of cerebellar hemangioblastoma using Onyx in the patient associated with multiple co-morbidities. MRI showed significantly decreased in size after embolization and demonstrated stable to minimally decreased size of the tumor after six months follow-up. However, by radiological and pathological assessment, there was

no necrosis of the whole tumor with always presenting of a rim of viable tissue assuming rarely curing of meningiomas by only embolization, and then definitive embolization should be considered for palliative shrinkage in debilitated patients⁽³⁴⁾. In the present study, after nearly complete devascularization of cerebellopontine angle (CPA) hemangioblastoma with NBCA, the patient (case 16) felt better and refused further surgery. Two months later, a follow-up MRI indicated tumor progression, and angiographic follow-up showed recruitment of new tumor feeder from ICA. This patient was operated on 171 days after embolization with less than 1,000 ml of blood loss (Fig. 6D, 6J, 6K). Therefore, when the tumor is decided

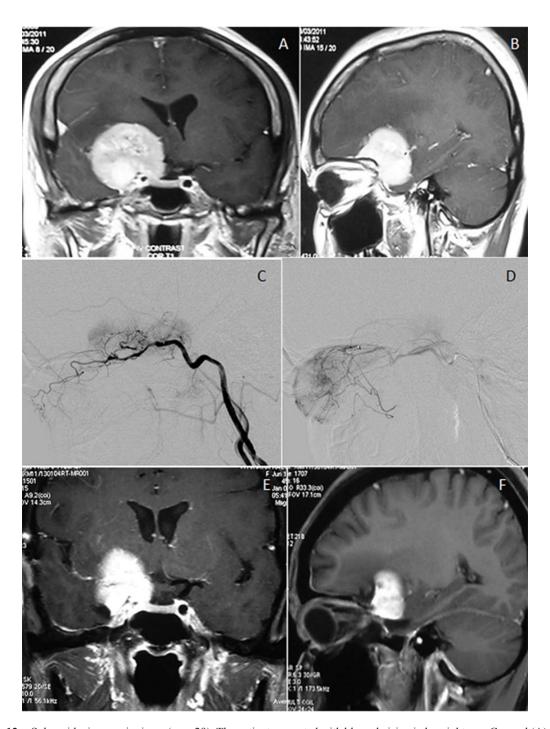


Fig. 12 Sphenoid wing meningioma (case 29): The patient presented with blurred vision in her right eye. Coronal (A) and Sagittal (B) T1-weighted Gd-enhanced MRI showed large homogeneously enhancing tumor causing pressure effect on right lateral ventricle, and encasement of the right ICA. (C), Lateral view of right CCA injection demonstrated the tumor stain fed mainly by branches of the right OphA. (D), The tip of microcatheter could not be placed into the proper position of the branch of the OphA. Pre-operative embolization was not performed in this patient. Two years after radiosurgery following subtotal tumor removal, Coronal (E) and sagittal (F) T1-weighted Gd-enhanced MRI revealed decreasing in tumor size and no pressure effect on the ventricle. Her vision has been fully recovered without new neurological deficits.

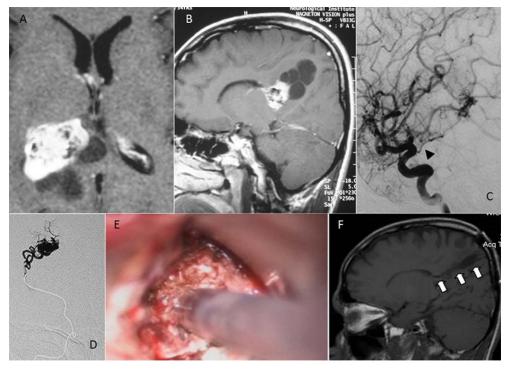


Fig. 13 Trigonal meningioma (case 37): Axial (A) and sagittal (B) T1-weighted Gd-enhanced MRI showed heterogeneously enhancing tumor in right trigone. Of note, the tumor was surrounded by lobulated cyst. (C), Lateral view of right ICA injection demonstrated tumor blush supplied mainly by the right AChA (arrowhead). (D), NBCA mixed with lipiodol was injected via the right AChA. (E), The tumor was removed with CUSA (Cavitron ultrasonic surgical aspirator) under microscope. (F), Post-operative MRI revealed surgical trajectory (white arrows) through parietal transcortical approach and disappearance of the tumor.

to be performed palliative embolization, a follow-up MRI is essential to visualize tumor regrowth, and cerebral angiography may be required to detect early revascularization. Re-embolization or surgery of the regrown tumor may be considered by individual neurosurgeon. Furthermore, radiosurgery after embolization should be used as another option in treatment of meningiomas⁽¹³⁾.

In contrast to the study by Kim et al⁽¹¹⁾ and Bendszus et al⁽¹⁶⁾ that reported no correlation between intra-operative blood loss and the angiographic extent of devascularization. The results of the present study showed that intra-operative blood loss was less in extensive or complete angiographic devascularization. However, Bendszus et al⁽¹⁶⁾ reported that there was correlation between intra-operative blood loss and the extent of non-enhancing areas on MRI after embolization. The effectiveness in reducing blood loss occurred when there was more than 90% tumor devscularization on post-embolization MRI. Grand et al⁽⁶⁾ also found that gadolinium (Gd)-enhanced MRI

is an excellent modality for evaluating the efficacy of an embolization. They suggested that the main reason of the discrepancy between the post-embolization angiography and the surgical findings was proximal spasm of feeding vessels. In the present study, the authors could not evaluate correlation between blood loss and the extent of non-enhancing areas on MRI because a follow-up MRI after embolization was obtained only in a small number of patients (Fig. 7D, 9J).

The presence of any early draining veins on angiography was used as one factor to assess the vascularity of the tumor⁽³⁴⁾. The early filling vein indicates an arteriovenous shunt and was initially understood as the angiographic sign of malignant brain tumor. Later, this angiographic pattern was reported in meningioma and hypervascular vestibular schwannoma^(60,61). An angiographic characteristic of hemangioblastomas also demonstrates significant vascular blush with arteriovenous shunting⁽⁴¹⁾. Few studies have shown that early draining veins carried

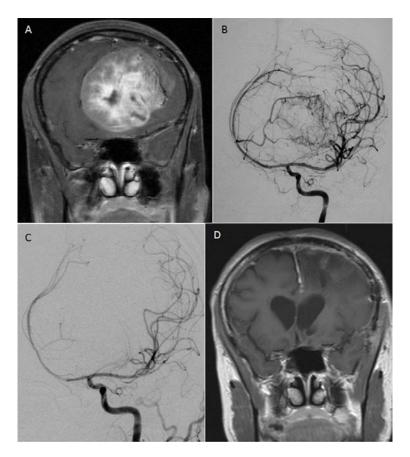


Fig. 14 Falcine meningioma (case 15): (A), Pre-operative T1-weighted Gd-enhanced coronal MRI showed large heterogeneously enhancing tumor with significant mass effect to frontal lobes. (B), Left ICA injection, AP view, demonstrated tumor stain supplied mainly by left pericallosal artery. (C), Complete devascularization of tumor was achieved. (D), Postoperative T1-weighted Gd-enhanced coronal MRI confirmed total resection of the tumor.

arterialized blood and made difficult hemostasis in piecemeal resection or internal decompression of the tumor, which needed to be managed by multi-staged operations^(62,63). Early appearance of draining veins in this present study was visualized not only in malignant tumors (i.e., GBM, metastasis, and hemangiopericytomas) but also in benign tumors (i.e., hemangioblastomas, meningiomas, glomus jugulare, mixed oligoastrocytoma, osteoblastoma, and neurofibroma) (Fig. 2F, 3E, 4G, 8C, 9F, 15C). Interestingly, the presence of early draining veins in meningiomas was frequently found in a transitional type, and not always in an angiomatous type. As mentioned earlier, NBCA is suitable for high flow fistula. Tumor with early filling of draining veins should then be embolized with NBCA.

Some studies^(5,24,50,54,64) reported the role of pre-operative embolization in tumors which were initially attempted to remove but had to be abandoned

because of uncontrollable intra-operative blood loss. In the present study, 12 of 37 patients (32%) had history of previous excessive bleeding during resection of tumors. These types of tumors include four hemangioblastomas, three hemangiopericytomas, three meningiomas, one glomus jugulare, and one neurofibroma. Interestingly, ten of 12 tumors had early draining veins detected on the angiographic studies. To reduce rates of uncontrollable bleeding during surgery, pre-operative identification of hypervascular tumors is important. Teasdale et al⁽³⁴⁾ sought a noninvasive method to select patients suitable for embolization. They found that CT and dynamic radioisotope scan findings were unable to predict the degree of vascularity of the tumor, and only angiography provided the necessary information. Nishimura et al⁽⁶⁵⁾ compared between 4D contrastenhanced MR angiography (4D-CE-MRA) at three tesla (3T) and DSA in evaluation of tumors of the head, neck

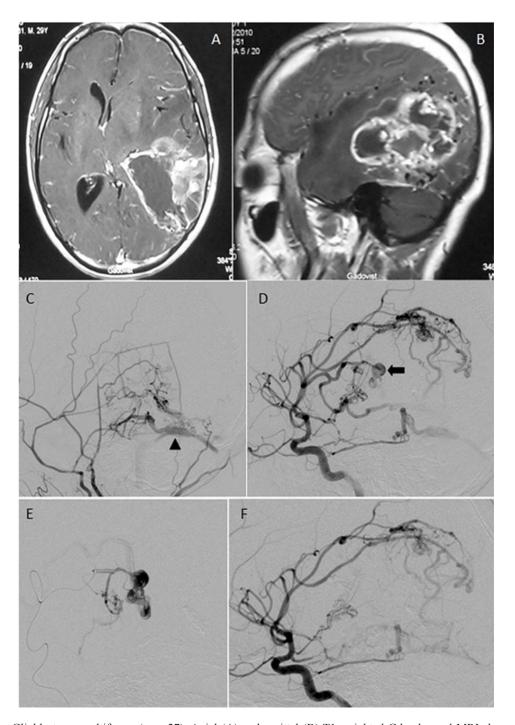


Fig. 15 Glioblastoma multiforme (case 27): Axial (A) and sagittal (B) T1-weighted Gd-enhanced MRI showed large irregular rim enhancing tumor in left temporo-occipital lobe. (C), Lateral view of left external carotid artery injection demonstrated multiple arteriovenous shunts fed by branches of MMA, STA, and OA with venous draining into left transverse sinus (arrowhead). (D), Lateral view of left ICA illustrated multiple tumor blushes and arteriovenous shunts supplied by angular branches and posterior temporal branches of MCA, AChA, and tentorial branch of MHT. Of note, intratumoral venous aneurysm is seen (the arrow). (E), Superselective angiography of left posterior temporal branch before embolization revealed the venous aneurysm and arteriovenous shunt. (F), Post-embolization angiography confirmed disappearance of the venous aneurysm.

and brain. They found that 4D-CE-MRA may be equivalent to DSA in identifying the presence or absence of hypervascular tumors, but it is not able to replace DSA in the planning of interventional procedures due to poor identification of feeding arteries. Another study from the same center by Uetani et al⁽⁶⁶⁾ compared between unenhanced 3D time of flight (TOF) MRA at 3T and DSA for identification of feeding arteries of intracranial meningiomas, and found that unenhanced 3D TOF MRA is a reliable diagnostic tool for identification the main arterial feeders. However, it cannot replace DSA for preinterventional evaluation because its identification of other feeders is relatively limited. In a large series of meningiomas by Waldron et al⁽²¹⁾, most patients were selected for possible embolization by evaluation of arterial supply from angiography. Wang et al⁽¹⁴⁾ diagnosed hypervascular brain tumors by using MRI findings of strong enhancement with presence of multiple dilated tumor vessels, and selected patients for pre-operative embolization using angiographic evaluation by finding at least one visible major feeding vessel.

Convexity meningiomas can usually be devascularized easily in the initial surgical exposure by occlusion of feeding vessels from external carotid branches. The main reason in the present study for pre-operative embolization of convexity tumors and other locations was MRI findings of numerous flow voids in and around the tumor representing hypervascular tumor (Fig. 2C, 2D, 4A, 4B, 4C, 6A, 10C, 11A, 11B). The present study showed that some convexity meningiomas not only fed by external carotid branches, but also supplied dominantly by pial branches of ICA (Fig. 4D, 4H, 4I).

Hemangiopericytomas are malignant extraaxial hypervascular tumors associated with large amounts of blood loss intra-operatively^(7,67,68). It is difficult to distinguish between meningiomas and hemangiopericytomas on MRI or CT scan⁽⁶⁸⁾. Marc et al⁽⁶⁹⁾ described multiple tiny irregular feeding vessels springing from a main trunk (i.e., corkscrew vessels) as a characteristic finding of hemangiopericytomas that found in every case, whereas few studies(67,68) rarely reported these abnormal vessels. The present study also found corkscrew vessels in only one in five patients (Fig. 3G). Contrary to a study by Marc et al⁽⁶⁹⁾, which showed no early draining vein in the region of the tumor, early draining vein was found in three out of five patients (Fig. 3E). Chiechi et al⁽⁷⁰⁾ studied in 34 pathologically proved cases of hemangiopericytoma, which MRI was performed in 18 cases, and found that

MRI of all 18 hemangiopericytomas showed prominent internal flow voids. Later, Akiyama et al⁽⁶⁷⁾ also reported prominent internal flow voids on MRI in most of patients with intracranial hemangiopericytomas, which had substantial intra-operative blood loss. Furthermore, vessels of hemangiopericytomas have thin walls and are difficult to coagulate by a bipolar device. Therefore, pre-operative embolization of these tumors is effective and can reduce requiring extensive blood transfusions⁽⁵⁴⁾. The major blood supply of these tumors arises from pial branches of ICA or VB system rather than from meningeal branches of ECA⁽⁶⁹⁾. Then, preoperative embolization of only ECA does not control the intra-operative bleeding. For example, in this present study, presurgical embolization of a huge recurrent skull base hemangiopericytoma was performed successfully via branches of both ECA and ICA (Fig. 3H). This was similar to a prior study by Kim et al⁽¹¹⁾, which used NBCA for embolization of these tumors.

In the present study, pre-operative embolizations were used in the deep-seated hypervascular brain tumors, that feeding vessels cannot be easily interrupted in the early stages of operation, including intraventricular, and skull base tumors (Fig. 3B, 3C, 3D, 7A, 12A, 12B, 13A, 13B). After embolization, the tumor could be removed by a piecemeal technique with ultrasonic aspirator (Fig. 13E). A comparative study in pre-operative embolization of intraventricular tumors (i,e., choroid plexus tumors) with NBCA by Haliasos et al⁽²⁴⁾, found that embolized group was resulted in significant reduction of perioperative blood loss, increasing rate of gross total removal, and decreasing the chances of abandoning the operation due to excessive bleeding. However, the concept of cranial base surgery was believed on early interruption of the tumor blood supply by cranial base surgical techniques should obviate the need for presurgical embolization in large skull base tumors, e.g., sphenocavernous meningiomas⁽⁷¹⁾. Embolization of meningiomas located in tuberculum sella or olfactory groove, which is typically supplied by the ethmoidal vessels from the OphAs, was also not recommended due to early access of feeding vessels in surgery and risk of vision(21).

Paragangliomas are highly vascular and usually benign, but locally infiltrating⁽²³⁾. Massive intra-operative bleeding during surgical resection of these tumors may be a major problem, especially when they are large⁽⁴⁵⁾. A comparative study of neck paragangliomas by Tikkakoski et al⁽⁵⁷⁾ concluded that the blood loss and the surgical time decreased

significantly in the embolized group. Pre-operative embolization of paragangliomas of the temporal bone should be performed in large glomus jugulare tumors involving the carotid canal, either with or without intracranial extension⁽²³⁾. Glomus jugulare tumors were supplied by external carotid branches (e.g., APhA, OA, MMA, posterior auricular artery (PAA)), internal carotid branches (e.g., ILT, caroticotympanic artery), and/or VB branches (e.g., AICA, PICA)(23,45). In the present study, a 51-year-old female with an extensive glomus jugulare tumor (case 8) underwent ineffective presurgical devascularization owing to prior ligation of the ipsilateral external carotid artery (Fig. 2G) and increasing tortuosity of collateral feeding vessels (Fig. 2I, 2J, 2K, 2L). This tumor was removed sub-totally with substantial intra-operative blood loss. Based on the technique described in previous studies(44,45), multiple direct percutaneous intratumoral injections with NBCA were performed to reduce volume of the tumor (Fig. 2N, 2O), and the patient was sent for further radiosurgery to control the residual tumor. The tumor was considered to be controlled (Fig. 2P) and the patient has been able to perform activities of daily living. Another patient (case 1) with large hemangiopericytoma also underwent ligation of one side of ECA like in the patient with paraganglioma. Prior ligation, ECA had increased the supply from contralateral side (Fig. 3E, 3F). This situation caused the embolization of the tumors more complicated because it was difficult to navigate the microcatheter through the tortuous vessels. Therefore, severe tortuosity of feedings vessels may preclude successful embolization.

Compared to presurgical embolization of extra-axial brain tumors, presurgical embolization of intra-axial brain tumors is performed much less due to technical difficulties and safety concerns⁽¹⁴⁾. Intra-axial brain tumors, which were embolized in the present study, include hemangioblastoma, GBM, and oligoastrocytoma. Few studies^(11,14,19) reported embolization of GBM, astrocytoma, and brain metastasis, whilst embolization of hemangioblastomas was the most commonly reported in many studies^(8,11,14,17,31,39,41,43,64). Similar to previous studies^(11,14), the present study preferred to use NBCA as the embolic material of choice for intra-axial gliomas.

Hemangioblastomas are benign intra-axial hypervascular tumors, commonly located in the cerebellum. They frequently appear in mixed solid and cystic components⁽⁷²⁾. Their blood supply are derived mainly from the SCA, AICA, and/or PICA. Hemangioblastomas, especially in recurrent tumors,

may also recruit blood vessels from dural arteries, including the MMA, the transmastoid branch of the OA, the neuromeningeal division of the APhA, the posterior meningeal branch of VA, and the tentorial artery of ICA(8,43,64). Treatment of these tumors, especially for the lesion with significant solid components, is often complicated due to excessive bleeding, which can obscure the operative field and promote damage to the surrounding tissue (39,43). Therefore, pre-operative embolization has a role in the resection of hemangioblastomas, especially in a large size(43). Cornelius et al(64) reported tumor bleeding and death in all three cerebellar hemangioblastomas after embolization with smaller particles. They reviewed the literature about particle embolization of hemangioblastomas and suspected that the use of smaller particles was a cause of venous congestion with subsequent congestion of tumor vessels, whereas all other authors who used larger particles led to the complete obliteration of tumor supply without hemorrhagic complications^(8,39,43). Gore et al⁽¹⁷⁾ initially reported successful pre-operative embolization of the cerebellar hemangioblastoma using combination of Onyx and NBCA in five arteries. Later, Horvathy et al⁽⁴¹⁾ achieved complete angiographic devascularization in a hemangioblastoma using Onyx, but microcatheter entrapment occurred and then was removed in a delayed fashion several hours after the procedure. Dabus et al⁽³¹⁾ also reported successful embolization of three hemangioblastomas using Oynx with transient symptoms from a small lateral medullary infarction in one patient. Similar to some studies(11,14), the authors of this present study prefer to use NBCA for presurgical embolization of hemangioblastomas, which are the most common intra-axial tumors in this study, via pial branches without any complication (Fig. 6G, 6H).

Pre-operative embolization may play a major role in tumors involving the scalp and calvarium because of significant bleeding just getting to the tumor⁽⁴⁶⁾. All three calvarial tumors and one tumor of the scalp in this present study were successfully embolized with NBCA without post-operative scalp necrosis (Fig. 9A, 9B, 9C, 9I).

In this present study, spontaneous intracranial hemorrhage associated with brain tumor occurred in three patients (Fig. 2A, 2B, 8A, 10A, 10B). The first patient (case 19) was a 34-year-old male with metaplastic meningioma at cribriform plate that cerebral angiography demonstrated the tumor stain with an early draining vein without any aneurysms (Fig. 8C), in which the mechanism of spontaneous hemorrhage in this case

remains unclear. The second patient (case 8) was a 51year-old female with glomus jugulare tumor that vertebral angiography revealed two AICA aneurysms, in which the proximal one caused hemorrhage. This proximal aneurysm was occluded with coiling and the distal one was obliterated with NBCA (Fig. 2E). The other patient (case 32) was a 67-year-old male with convexity meningioma that cerebral angiography showed no any aneurysms or early draining veins. A study of Ho⁽⁷³⁾ reviewed the literature of intracranial neoplastic aneurysms and found that metastatic tumors appeared to be more common cause of massive intracerebral hemorrhage than primary tumors by invasion of arterial wall including the elastic lamina by neoplastic cells. There was another report of neoplastic aneurysm associated with solitary fibrous tumor arising in orbit, anterior, and middle cranial fossa⁽¹⁹⁾. Another patient (case 27), with unruptured intra-tumoral aneurysm in this present study, was a 29-year-old male with GBM that selective angiography showed the venous aneurysm arising from posterior temporal branch of the left MCA (Fig. 15D, 15E). This aneurysm was occluded with NBCA to prevent massive hemorrhage during surgery.

The review of the literature by Kallmes et al⁽⁵⁰⁾ about hemorrhagic complications in embolization of meningiomas found that most hemorrhages occurred in large tumors immediately after complete devascularization with small or intermediate size of particles via the MMA. Wang et al⁽¹⁴⁾ hypothesized two probable mechanisms of hemorrhage after particle embolization. Firstly, delayed hemorrhage may have been caused by rupture of some patent collateral tumor vessels after occluding part of the terminal tumor arteries. Secondly, increasing pressure from continuous injection of the particles into a wedged catheter in feeding artery may cause periprocedural hemorrhage. They also suggested another two causes of periprocedural tumor hemorrhage, including forceful manipulation of microcatheter and micro-guidewire injuring the fragile tumor vessels and forceful test injection from the microcatheter causing rupture of the tumor vessels. In the present study, extravasation of contrast material occurred during embolization in a 58-year-old female (case 18) with a large falcine meningioma due to manipulation of microcatheter and micro-guidewire in the fragile pial branch. Embolization with NBCA was rapidly performed to achieve adequate sealing for this defect (Fig. 5E, 5F, 5G). The patient underwent successful embolization and subsequent surgery with uneventful recovery one

week later. Identifying hemorrhagic complication during embolization and promptly performing appropriate management are critical to improve the outcome of the patient.

The STA and OA provide the major arterial supply to the scalp, thus inadvertent occlusion of normal branches of these vessels may lead to ischemic necrosis of the scalp. Several studies(4,34,74,75) reported post-operative scalp necrosis in patients, which most of them required reconstructive surgery by plastic surgeon, underwent pre-operative embolization through branches of ECA, especially in STA or OA. During sub-selective catheterization of feeding vessels, the tip of microcatheter should be placed as close to the tumor as possible to avoid reflux of embolic materials to normal adjacent tissues, especially skin. Macpherson⁽⁴⁾ advocated temporary occlusion of STA during the injection of embolic materials to prevent emboli moving to the scalp. The scalp flap must be designed carefully to avoid further disruption of compromised arterial supply, when the major branches of ECA are occluded^(74,75). Ischemic scar in the patient (case 32), who had scalp necrosis after embolization through STA with subsequent surgery in this present study, resolved spontaneously without surgical intervention (Fig. 10H).

The limitations of the present study include small sample size, a retrospective design, and a non-comparative study. Therefore, a prospective, randomized controlled study with larger sample size should be conducted in the future. Additionally, MRI studies should be obtained to assess tumor necrosis following embolization in all patients for evaluating the effectiveness of embolization. This present study did not evaluate histological evidence of tumor necrosis after embolization from pathological studies, however, previous studies^(3,34) reported discrepancies between CT scans after embolization and pathological findings. The major cause of difficulties in assessment of the effects of embolization was the specimens consisted of multiple small fragments rather than an intact tumor.

Conclusion

Although pre-operative embolization of intraand extracranial tumors was safe, only extensive or complete angiographic devascularization has been effective in less intra-operative blood loss. Indications for pre-operative embolization include: a history of excessive bleeding from previous surgery; known hypervascular tumor types (e.g., hemangiopericytoma, hemangioblastoma, paraganglioma); the presence of multiple flow voids on MRI; hypervascular tumors of skull or scalp; deep-seated tumors (e.g., cranial base tumor, intraventricular tumor) with difficulties in early surgical access of the main feeding vessels, and tumors associated with intratumoral aneurysm. Timing to surgery after embolization depends on embolic material chosen. Early subsequent surgery should be considered in very large tumors with midline shift or hydrocephalus. Under sub-selective catheterization, NBCA was used for the pial branches of ICA and VB system, and PVA particles and/or NBCA for the branches of ECA. Importantly, knowledge of angioarchitecture or vascular anatomy of the tumors is fundamental for safe and effective embolization.

What is already known on this topic?

Pre-operative embolization of meningiomas has been widely used to reduce blood loss during surgery, whereas pre-operative embolization of non-meningiomas is much less performed in many institutes. However, the decision of using this procedure depends on the personal preference of the neurosurgeon. Indications of presurgical embolization of meningiomas and non-meningiomas remain unclear. PVA particles have been the most commonly used for embolization of meningiomas through external carotid branches. Subsequent surgery after embolization is usually performed within a week.

What this study adds?

The authors examined pre-operative embolization in both meningiomas and non-meningiomas simultaneously because they have the same pattern of vascular supply arising from ICA, ECA, and/or VB system depending on their locations. Indications of this procedure were reviewed and proposed. Embolization via pial branches of ICA or VB system can be performed safely with NBCA. Timing to surgery after embolization depends on embolic material chosen. Definitive embolization of brain tumors should be considered for palliative therapy in inoperable patients. Only extensive or complete angiographic devascularization has shown the achievement of reduction intra-operative blood loss.

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Potential conflicts of interest

None.

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การอุดหลอดเลือดที่ไปเลี้ยงเนื้องอกที่อยู่ภายในและภายนอกกะโหลกศีรษะก่อนการผ่าตัด: ข้อมูลจากผู้ป่วย 37 ราย

ประเสริฐ เอี่ยมปรีชากุล, วุฒิพงษ์ ฐิรโฆไท, ปัญจมา เลิศบุษยานุกูล, สมเกียรติ ศิริวิมลมาส, อนุศักดิ์ เลียงอุดม

วัตถุประสงค์: เพื่อศึกษาความปลอดภัยและประสิทธิภาพของการอุดหลอดเลือดที่ไปเลี้ยงเนื้องอกที่อยู่ภายในและภายนอกกะโหลกศีรษะ ก[่]อนการผาตัด และเพื่อที่จะหาขอบงชี้ในการเลือกผู*้*ป่วยมาทำหัตถการนี้

วัสดุและวิธีการ: เป็นการศึกษารวบรวมข้อมูลแบบย้อนหลังในช่วงเวลาระหว่างเดือนมิถุนายน พ.ศ. 2551 ถึง สิงหาคม พ.ศ. 2555 ของผู้ป่วย 37 ราย (ผู้ชาย 17 ราย ผู้หญิง 20 ราย อายุเฉลี่ย 44.2±14.2 ปี) ที่ได้รับการอุดหลอดเลือดที่ไปเลี้ยงเนื้องอกที่อยู่ภายในและภายนอกกะโหลกศีรษะ ก่อนการผ่าตัดโดยประเมินจากลักษณะเฉพาะของเนื้องอก (ชนิด, ตำแหน่ง, ปริมาตร, เปอร์เซ็นตของหลอดเลือดที่มาเลี้ยงเนื้องอก, การพบ early draining vein), ขอบเขตของการขาดเลือดของเนื้องอกจากภาพถ่ายรังสีหลอดเลือด, ช่วงเวลาระหว่างการอุดหลอดเลือดไปจนถึงวันผ่าตัด, ปริมาณเลือด ที่เสียไประหว่างผ่าตัด และภาวะแทรกซอนที่เกี่ยวข้องกับการอุดหลอดเลือด

ผลการศึกษา: จากการศึกษาพบเนื้องอก 37 ราย (ปริมาตรเฉลี่ย 90.9±83.6 ลูกบาศก์เซนติเมตร) ประกอบด้วยชนิด meningioma 18 ราย, hemangioblastomas 6 ราย, hemangiopericytomas 5 ราย, metastasis 1 ราย, osteoblastoma 1 ราย, osteosacroma 1 ราย, neurofibroma 1 ราย, central neurocytoma 1 ราย, glomus jugulare 1 ราย, mixed oligoastrocytoma 1 ราย และ glioblastoma multiforme 1 ราย early draining vein พบในผู้ป่วย 24 ราย (64.9%) ความล้มเหลวของการอุดหลอดเลือดที่ไปเลี้ยงเนื้องอกก่อนการผาตัดเกิดขึ้นกับผู้ป่วย 4 ราย ระยะเวลาเฉลี่ยหลังจากอุดหลอดเลือดไปจนถึงผาตัดเท่ากับ 7 วัน (พิสัย 1-171 วัน) พบความแตกตางกันอย่างมีนัยสำคัญทางสถิติระหวางระดับ การขาดเลือดของเนื้องอกจากภาพถ่ายรังสีหลอดเลือดกับปริมาณเลือดที่เสียไประหวางผาตัด (p = 0.009, Kruskal-Wallis test) ผู้ป่วย 2 ราย (5.4%) เกิดภาวะแทรกซ้อนที่เกี่ยวข้องกับการอุดหลอดเลือด 1 ราย มีเลือดออกชวงระหวางกำลังใส่สายสวนขนาดเล็กเพื่อเข้าไปในหลอดเลือด ที่เลี้ยงเนื้องอกอีก 1 รายมีหนังศีรษะขาดเลือดหลังผาตัด

สรุป: ถึงแม้ว่าการอุดหลอดเลือดที่ไปเลี้ยงเนื้องอกที่อยู่ภายในและภายนอกกะโหลกศีรษะก่อนการผ่าตัดจะมีความปลอดภัย แต่จะมีประสิทธิภาพ ในการลดการเสียเลือดในระหว่างผ่าตัดได้ก็ต่อเมื่อสามารถอุดหลอดเลือด ที่ไปเลี้ยงเนื้องอกได้ในระดับที่ค่อนข้างมาก หรืออย่างสมบูรณ์จากการศึกษานี้ พบว่าข้อบ่งชี้ในการทำหัดถการนี้ประกอบด้วยการมีประวัติสูญเสียเลือดอย่างมากจากการผ่าตัดครั้งก่อนหน้านี้, ทราบอยู่แล้วว่าเป็นเนื้องอกชนิดที่มี หลอดเลือดมาเลี้ยงมากมาย (เช่น hemangiopericytoma, hemangioblastoma และ paraganglioma), การพบ multiple flow voids บนภาพถ่าย MRI, เนื้องอกชนิดที่มีหลอดเลือดมาเลี้ยงมากมายของกะโหลกศีรษะหรือหนังศีรษะ, เนื้องอกที่อยู่ในตำแหน่งลึกยากต่อการเข้าถึงหลอดเลือดที่มาเลี้ยง เนื้องอกในช่วงแรกของการผ่าตัด (เช่นเนื้องอกบริเวณฐานกะโหลกและเนื้องอกภายในโพรงสมอง), และเนื้องอกที่มีหลอดเลือดโปงพอง (aneurysm) ภายในเนื้องอก