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Extended Preoperative Polyvinyl Alcohol Microembolization of Intracranial Meningiomas: Assessment of Two Embolization Techniques

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PURPOSE: To evaluate the efficacy of preoperative meningioma devascularization with small polyvinyl alcohol (PVA) particles. **METHODS:** In 34 patients with intracranial meningiomas, CT, MR, ^{1}H MR spectroscopy, MR volumetric measurements, intraoperative ultrasound, and histopathologic findings were used to compare the efficacy of two embolization techniques: 1) administration of 150- to 300-μm PVA particles in the usual suspension, and 2) administration of 50- to 150-μm PVA particles in a highly diluted suspension. **RESULTS:** Angiography after embolization demonstrated the total elimination of tumor blush in all patients. Contrast-enhanced MR after the administration of 150- to 300-μm PVA particles revealed a reduction of tumor enhancement in only two out of 14 patients. Only after the use of small particles could significant tumor necrosis be depicted on MR and confirmed histopathologically after surgery. In 12 of 20 patients, 30% to 95% of the whole tumor was necrotic with 17% to 20% reduction of tumor volume in four cases, leading to recovery from the initial neurologic deficits. In three of 20 patients without sufficient steroid medication before the treatment, tumor swelling occurred. Postembolization MR disclosed a tumor volume increase of 10% to 20% in these patients. ^{1}H MR spectroscopy of the tumors showed an increase of lactate and aliphatic lipid compounds after embolization, indicating tumor infarction. Surgical removal of effectively embolized meningiomas without significant blood loss was possible. The appearance of the tumor at operation, ultrasound examination, and the histopathologic examination of different parts of the tumor confirmed the preoperative MR findings suggesting necrosis. **CONCLUSION:** Extended microembolization with 50- to 150-μm PVA particles improves the surgical treatment of meningiomas, as compared with larger particle embolization. It may also be the only treatment required in older or high-risk patients. The protective effect of steroid medication before the endovascular treatment of meningiomas is suggested by our study.

Index terms: Meninges, neoplasms; Interventional materials, particles and microspheres; Interventional materials, embolic agents

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Preoperative embolization of intracranial meningiomas is a well-established procedure for reducing intraoperative blood loss. Attempts at devascularization of tumor tissue using different embolic materials, such as Gelfoam, lyophilized Dura, butyl-2-cyanoacrylate (NBCA), silastic spheres, polyvinyl alcohol (PVA) particles, and liquid embolic material (1–9) have been made with varying results. However, intraoperative and histologic findings have not generally confirmed extensive tumor necrosis, even in cases in which the angiogram showed complete devascularization (3, 6, 8, 9). This study focuses on the technique of PVA particle embolization, particularly the effect of particle size on tumor devascularization and tissue necrosis, as well as ease of resection. The value of this improved embolization technique as the sole treatment of meningi-

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omas in high-risk patients and for tumors supplied exclusively by the external carotid artery will be discussed.

Patients, Materials, and Methods

Technical Procedure

Superselective embolization of meningiomas was undertaken in 34 patients (24 women and 10 men; mean age, 60; range, 25 to 80 years). Diagnostic angiography and preoperative embolization were carried out at the same session under local anesthesia using a digital subtraction angiography unit and the transfemoral approach. Clinical observation of the patient was possible throughout the procedure. A microcatheter system (Tracker 18 or Tracker 10, Target Therapeutics, San Jose, CA) was introduced into the main feeding vessels which in most cases arose from the external carotid artery. Two cases involved feeding arteries arising from the tentorial artery. Embolization was performed after superselective catheterization of the tentorial artery in one patient. In the other patient, temporary balloon occlusion of the internal carotid artery (ICA) was performed and PVA particles were then injected into the ICA proximal to the temporary occlusion (10). After complete obliteration of the tumor-feeding vessels, the ICA was extensively flushed with saline through a 5-F diagnostic catheter to avoid occlusion of normal intracranial vessels by any PVA particles that might have remained proximal to the inflated balloon (Figs. 1A and 1B). Before the inflation of the balloon, 7000 IU of heparin was given intravenously and clinical tolerance of the occlusion assessed over a period of 20 minutes.Tumor embolization was carried out with PVA microparticles, using two different techniques:

Protocol 1

The conventional technique, as previously described (6, 10), was used on 14 patients. PVA particles of 150- to 300-μm in a suspension of 0.4 gm/250 mL saline were injected through the microcatheter until angiographic disappearance of the tumor blush was achieved. This procedure was repeated, whenever possible, for all main vessels supplying the tumor. The average embolization time for each vessel was 20 to 30 minutes.

Protocol 2

The above-mentioned technique with the modifications described below was used on 20 patients, with the goal of increasing the efficacy of the embolization. The particle size was reduced to 50- to 150-μm and 0.1 gm of PVA particles were suspended in 1000 mL of saline. After sedimentation of the larger particles, mostly 50-μm particles were left in suspension, as ascertained by cell-counter analysis. These small particles were injected in highly diluted suspension. The injection of PVA particles was carried out extremely slowly, allowing the blood to carry the particles as far distally as possible. The typical embolization time was 60 to 120 minutes for each feeding vessel and even as long as 150 minutes in extensively vascularized tumors. The progressive centripetal obliteration of tumor vessels (Figs. 2A–2D) causes an increase in peripheral vascular resistance. This eventually results in a marked increase in the force needed for injection, which is an indication for control angiography and often for the cessation of the procedure.

Diagnostic Assessment

Imaging

Both magnetic resonance (MR) and computed tomography (CT) were performed within 2 days before embolization, and repeated 4 to 6 days afterwards. In five patients, the postembolization imaging was carried out after an interval of 4 to 6 hours.

MR was performed with a 2-T whole-body system (Bruker S 200). T2-weighted axial 5-mm spin-echo (SE) images (2450/32/2) (TR/TE/excitations) and T1-weighted 5-mm or 3-mm axial, coronal, and sagittal SE sequences (500/20/2) were obtained. The latter sequence was repeated after bolus injection of paramagnetic contrast material (Gd-DTPA, 0.1 mmol/kg).

Volumetric Measurements

Computer-aided volumetric measurements, based on the MR data obtained in three planes, allowed changes in tumor volume to be observed with a high degree of accuracy (11, 12). The total tumor volume was calculated from the MR scans before and after the embolization. The calculations were based on T1-weighted contrast-enhanced images. Postembolization nonenhancing parts were considered as devascularized tumor regions.

^{1}H MR Spectroscopy

In 17 cases from group 2, localized ^{1}H MR spectroscopy was carried out 4 to 6 hours, 24 hours, and 4 days after embolization. These spectra were compared with the preembolization findings. In this way, the presence of lactate as a sign of tumor ischemia could be readily detected (13). Volume selection for single-voxel SE spectra (TE = 135 msec, TR = 1777 msec, 256 average, $(2\ cm)^3$) was based on a T2-weighted pilot scan, which was used to position the voxel in those parts of the tumor showing distinct changes after embolization. Further details will be published elsewhere.

Surgery

Surgery was performed, whenever possible, within 1 week after embolization (group 1: range, 1–14 days; mean, 8.2 days; group 2: range, 5–9 days; mean, 7.5 days). The microneurosurgical technique involved excavation of the central parts of the tumor mass, allowing the tumor to collapse and enabling easy dissection of the arachnoid

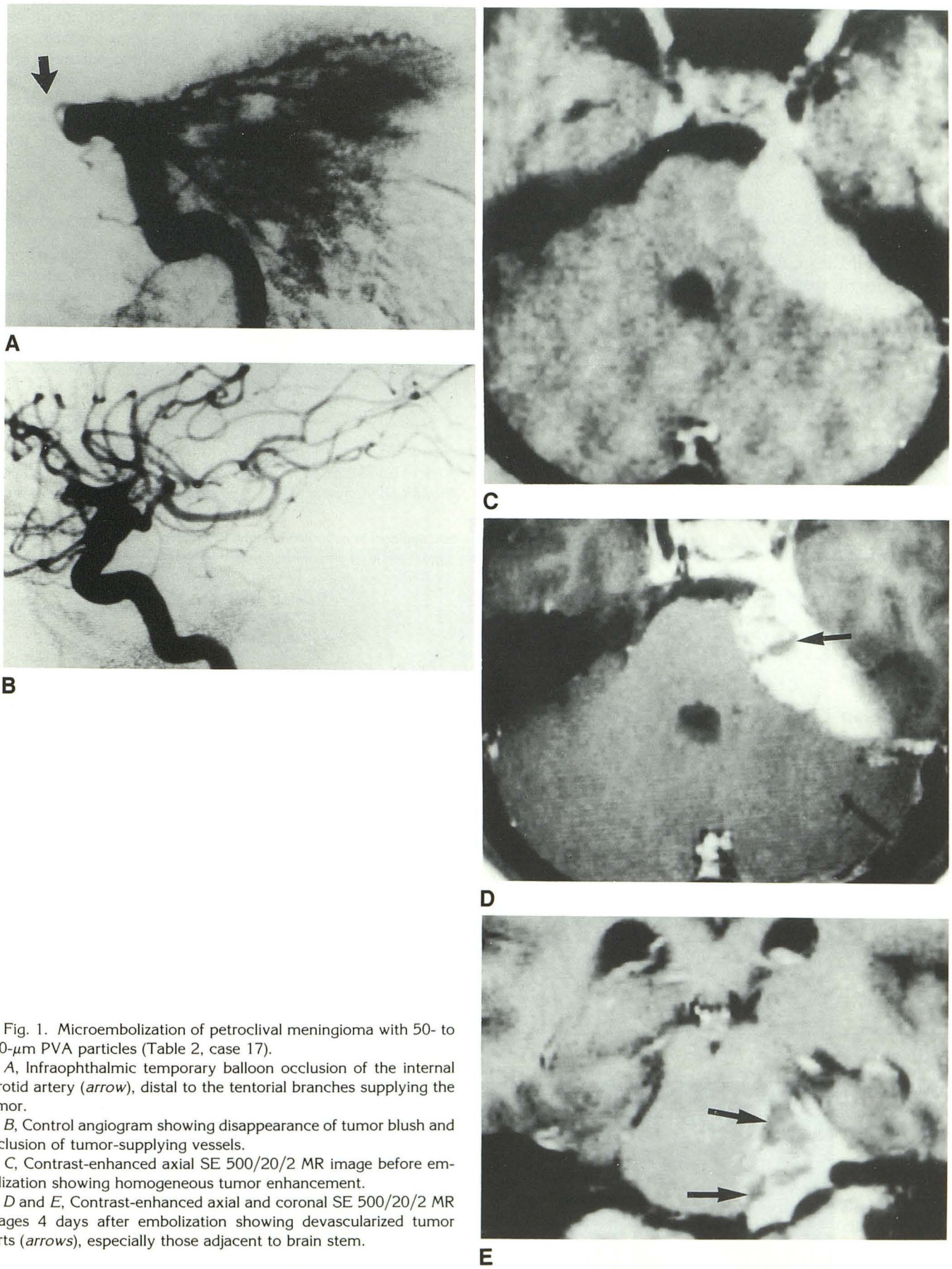


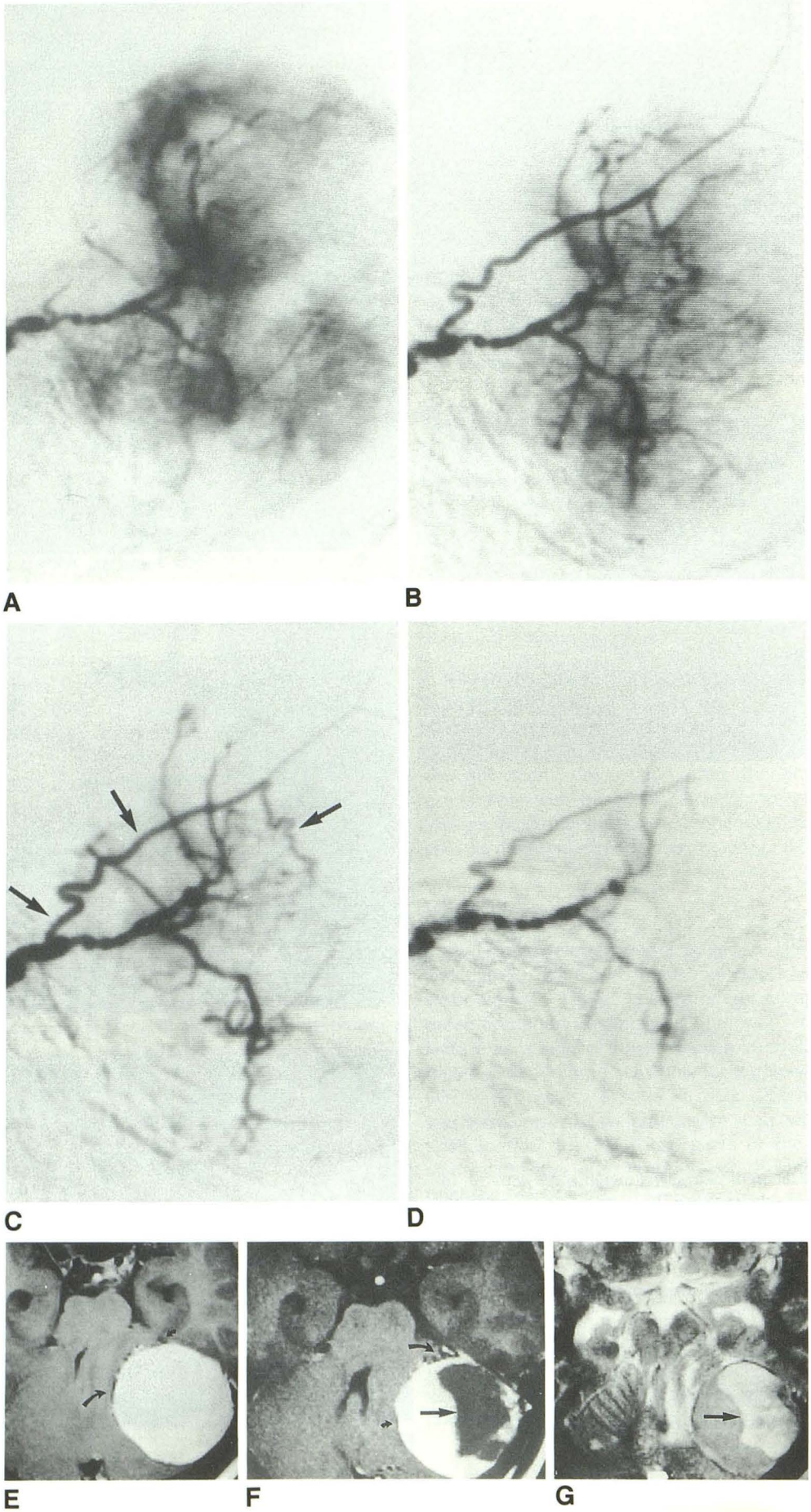
Fig. 1. Microembolization of petroclival meningioma with 50- to 150-μm PVA particles (Table 2, case 17).

A, Infraophthalmic temporary balloon occlusion of the internal carotid artery (*arrow*), distal to the tentorial branches supplying the tumor.

B, Control angiogram showing disappearance of tumor blush and occlusion of tumor-supplying vessels.

C, Contrast-enhanced axial SE 500/20/2 MR image before embolization showing homogeneous tumor enhancement.

D and *E*, Contrast-enhanced axial and coronal SE 500/20/2 MR images 4 days after embolization showing devascularized tumor parts (*arrows*), especially those adjacent to brain stem.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layers for total removal. The vascularity, consistency, and macroscopic appearance of the tumor were reported by the neurosurgeon. During the operation, ultrasound imaging (Ultramark 4, Advanced Technology Laboratories, Bothell, WA) was used by the neurosurgeon to locate biopsy sites in those parts of the tumor not enhancing on MR. The ultrasound equipment consisted of a sector probe, using frequencies of 5.0 MHz, 7.5 MHz, and 10 MHz.

Clinical Data

The clinical history and neurologic state before and after embolization and after operation were recorded. Patients were generally treated with dexamethasone (8 mg, 3 times a day) for at least 4 days before embolization, and up to 2 weeks after operation (initially 8 mg, 6 times a day, gradually reduced after 4 days).

Pathology

Biopsies were taken from specified areas of the tumor as described above and examined in a double-blind fashion by the neuropathologist after usual fixation and staining with hematoxylin eosin. Tibor-Pap and Van Gieson staining were also performed to identify embolization material within the tumor. A standard protocol was used to assess the extent of necrosis within each tumor specimen.

RESULTS

Group 1

Results of tumor embolization with 150- to 300-μm PVA particles in 14 cases are summarized in Table 1. Contrast-enhanced MR showed avascular regions within the tumor in only two patients (cases 4 and 12); these areas corresponded closely to nonenhancing areas in the postcontrast CT scans. MR volumetric measurements did not show any significant change in tumor volume or peritumoral edema after embolization in the patients of this group. Some decrease in the vascularity of the tumor was reported in most cases during operation. The estimated blood loss ranged from 50 to 200 mL

TABLE 1: Results of conventional microembolization of meningiomas using 150- to 300-μm PVA particles

Case No.	Sex, Age	Location	Embolized Vessels[a]	Necrosis in %[b]	Volume/ Change (MR)	Complications/Side Effects	Blood Loss at Surgery	Histology
1	F, 59	Middle cranial fossa	MMA		NS	Headache	2600 mL	Transitional
2	F, 79	Convexity	MMA + STA		NS	No side effects	500 mL	Syncytial
3	M, 41	Parasagittal-falx	MMA		NS	Headache	600 mL	Syncytial
4	M, 42	Middle cranial fossa	MMA	20	NS	No side effects	2000 mL	Syncytial/fibroblastic
5	M, 62	Middle cranial fossa	MMA		NS	No side effects	200 mL	Transitional
6	F, 67	Middle cranial fossa	MMA + MaxA		NS	Headache	700 mL	Syncytial
7	F, 72	Middle cranial fossa	MMA		NS	Headache	1500 mL	Transitional
8	F, 79	Convexity	MMA		NS	Acute bleeding; aphasia, hemiparesis[c]	1000 mL	Syncytial
9	F, 79	Petrous bone	MMA		NS	No side effects	50 mL	Syncytial/fibroblastic
10	F, 69	Convexity	MMA		NS	Headache	No surgery	No histology
11	F, 70	Middle cranial fossa	MMA		NS	Headache	700 mL	Fibroblastic
12	F, 69	Parasagittal-falx	MMA	40	NS	Headache	800 mL	Transitional
13	F, 74	Petrous bone	MMA		NS	No side effects	150 mL	Syncytial
14	F, 54	Middle cranial fossa	MMA + MaxA		NS	Facial pain	200 mL	Syncytial

[a] MMA = middle meningeal artery; ICA = internal carotid artery; MaxA = maxillary artery; STA = superficial temporal artery.

[b] MR finding, histologically confirmed.

[c] Complete recovery after surgery.

Fig. 2. Superselective microembolization of meningioma of the petrous bone with 50- to 150-μm PVA particles (Table 2, case 1).

A–D, Lateral subtraction angiogram of petrosquamosal branch of the middle meningeal artery. Preembolization tumor blush with tip of microcatheter placed close to tumor nidus (*A*). Control angiograms at 30-minute (*B*), 60-minute (*C*), and 90-minute (*D*) intervals after initiation of embolization showing gradual centripetal reduction of tumor blush. Note the collateral tumor-supplying vessel (*C*, *arrows*) showing prominent contrast after major reduction of peripheral tumor blush. Final postembolization angiogram (*D*) revealing occlusion of collateral branch with major tumor-supplying vessel still visible.

E, Contrast-enhanced axial SE 500/20/2 MR image before embolization showing homogeneous tumor enhancement. Dilated tumor-draining veins (*curved arrows*).

F and *G*, Postembolization contrast-enhanced axial SE 500/20/2 and T2-weighted 2450/32/2 images 4 days after treatment revealing devascularized tissue with increased water content (*straight arrow*) and partial collapse of tumor-draining veins (*curved arrows*). Note reduction of mass effect on adjacent brain tissue and fourth ventricle.

(four cases, or 30%) to 500 to 2600 ml (nine cases, or 70%). The clinical symptoms were not affected by embolization in 13 cases. However, in one case, a severe complication occurred. This patient (case 8) developed aphasia and a hemiparesis of grade 1–2/5 after embolization. CT revealed acute intratumoral bleeding. Surgery was undertaken immediately, and both the aphasia and the hemiparesis disappeared completely. One patient (case 10) with a meningioma of the convexity and nonspecific symptoms, such as dizziness, pain in the neck, and tiredness, refused surgery and was treated with embolization alone. The patient reported subjective improvement of dizziness after endovascular treatment. The long-term results have yet to be evaluated.

The histopathologic findings showed evidence of necrosis only in the nonenhancing areas of the embolized tumors on postcontrast MR/CT (cases 4 and 12). Necrosis was considered to be extensive in both cases, but vital cells were scattered within necrotic areas. Embolic material was found in the large vessels feeding the tumor. There was, however, no evidence of PVA particles in the precapillary tumor bed.

Group 2

A total of 20 patients were embolized with 50- to 150-μm PVA particles. The embolized vessels and results are summarized in Table 2. MR showed avascular regions within the tumor in all cases, ranging from 5% up to 95% of the total tumor volume. These areas corresponded to nonenhancing regions on contrast-enhanced CT scans. In the T2-weighted images a significant increase in the signal was seen in those regions of the tumor not enhancing on gadolinium-enhanced T1-weighted images. Volumetric measurements revealed a significant change in the tumor volume in seven patients, with an increase in volume of 10% to 25% in three patients, and a decrease in volume of 17% to 20% in four patients. Visible devascularization was reported by the surgeons in most cases as obvious pale areas in the central parts of the tumor during the operation (Fig. 3). In eight cases (40%), the tumor appeared to be macroscopically necrotic and of a fragile, partly liquid consistency. In case 12, residual vascularization of the tumor rim by cerebral pial arteries was confirmed during operation.

The postoperative clinical outcome was without major complications in all cases. All patients could be mobilized on the first postoperative day. The slight hemiparesis that occurred after embolization in patient 2 because of swelling of the tumor (Fig. 4) disappeared completely within 2 days of operation.

In patient 13, a 54-year-old woman with a meningioma of the convexity, who presented with brachial hemiparesis grade 2–3/5, seizures, persistent dizziness, lack of concentration, and memory disturbance, the symptoms disappeared within 2 days of embolization. Considering her clinical improvement with 95% devascularization and 20% reduction of the tumor volume after embolization, the neurosurgeons decided against operation. Four days after embolization, the patient was discharged; 1 week later, she was able to start work again for the first time in 6 months. Follow-up MR examinations after 3 and 9 months revealed a reduction in tumor size to only 36% and 30%, respectively, of the initial tumor volume. The long-term results are still awaited (Fig. 5).

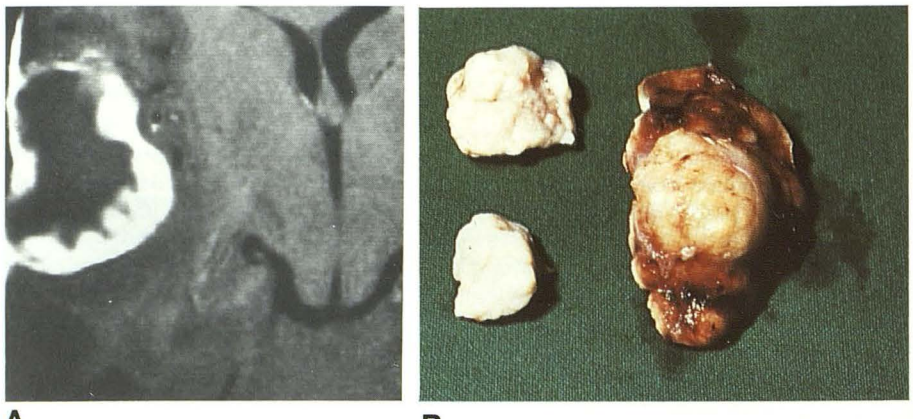

Fig. 3. Temporal convexity meningioma after microembolization with 50- to 150-μm PVA particles (Table 2, case 9).

A, Contrast-enhanced axial SE 500/20/2 MR images 4 days after embolization showing vascularized tumor tissue in the medial and lateral regions of the meningioma, while the central regions appear completely devascularized.

B, Macroscopic findings correlate well with the MR image with devascularized pale central tumor and partly vascularized lateral tumor adjacent to the dura.

TABLE 2: Results of extended microembolization of meningiomas with 50- to 150-μm PVA particles

Case No.	Sex, Age	Location	Embolized Vessels[a]	Necrosis in %[b]	Volume/ Change (MR)	Complications/Side Effects	Blood Loss at Surgery	Histology
1	F, 47	Petrous bone	MMA	50	NS	Headache	200 mL	Fibroblastic
2	F, 56	Convexity	MMA	70	+23%	Transient hemiparesis	500 mL	Fibroblastic
3	F, 42	Parasagittal-falx	MMA	30	NS	Headache	200 mL	Fibroblastic
4	M, 71	Convexity	MMA	40	NS	No side effects	500 mL	Fibroblastic
5	F, 62	Parasagittal-falx	MMA	20	+25%	No side effects	300 mL	Fibroblastic
6	F, 43	Petroclival	MMA + ICA	40	NS	Vomitus, headache	200 mL	Syncytial
7	F, 69	Anterior cranial fossa	MaxA	10	NS	No side effects		Syncytial
8	M, 67	Convexity	STA + MMA	5	+10%	No side effects	800 mL	Syncytial
9	F, 46	Middle cranial fossa	MMA	70	NS	Dizziness	500 mL	Fibroblastic
10	F, 80	Parasagittal-falx	MMA	5	NS	No side effects	500 mL	Angioblastic
11	M, 68	Parasagittal-falx	MMA + STA	50	NS	No side effects	800 mL	Syncytial
12	F, 66	Middle cranial fossa	MMA	20	−20%	Headache, facial pain	300 mL	Fibroblastic
13	F, 54	Convexity	MMA	95	−20%[c]	No side effects	No surgery performed	No histology
14	M, 47	Convexity	MMA	5	NS	Headache	500 mL	Transitional
15	M, 59	Middle cranial fossa	MMA	15	−17%	No side effects	250 mL	Syncytial
16	M, 25	Convexity	MMA	90	NS	No side effects	No blood loss	Fibroblastic
17	F, 58	Petroclival	ICA	10	NS	No side effects	150 mL	Syncytial/angioblastic
18	F, 58	Petrous bone	MMA	70	NS	No side effects	50 mL	Syncytial
19	M, 27	Convexity	MMA	90	−20%	Facial pain	No blood loss	Syncytial
20	F, 76	Parasagittal-falx	MMA	60	NS	No side effects	50 mL	Fibroblastic

[a] MMA = middle meningeal artery; ICA = internal carotid artery; MaxA = maxillary artery; STA = superficial temporal artery.
[b] MR finding, histologically confirmed; except for case 13 (no surgery carried out).
[c] Follow-up after 3 months: −64%.

Histopathologic Findings

All specimens of the tumor from nonenhancing parts on postcontrast MR showed extensive necrosis. Biopsies taken from enhancing parts frequently showed necrobiotic as well as vital cells. Embolic material was detected in 17 cases (85%) and reached the capillary bed in 15 cases (Fig. 6). In patients 8, 10, and 14, with tumors supplied mainly from the ICA, small necrotic areas without embolic material within the tumor were seen.

^1H MR Spectroscopy

Initial spectroscopy showed the typical spectral pattern for meningiomas described previously (14–16) in all patients. Four hours after embolization, a distinct lactate peak (at 1.3 ppm) was seen in those parts of the tumor showing changes in appearance on the T2-weighted image. Lactate was not seen on later spectroscopy, but a broad peak corresponding to aliphatic lipid compounds was present. An example of spectroscopic changes in tumor metabolism found after embolization is shown in Figure 7.

Discussion

The superselective preoperative embolization of meningiomas has been shown to be a great advantage in the devascularization of tumor tissue (3). Many authors (3, 6, 8, 9) using CT after embolization have described decreased contrast enhancement of the tumor mass. Hieshima et al (3) have observed a slight reduction in the tumor volume. However, no systematic histologic investigation, especially on the different contrast-enhancing parts of the tumor, has been undertaken to correlate the preoperative findings with the histopathology. Hence, the importance of preoperative embolization could be supported only by calling attention to the decreased intraoperative blood loss and the simplified removal of the tumor tissue.

Although a few authors have reported tumor necrosis after embolization by histology, the biopsies obtained during operation were not correlated with the postembolization CT or MR findings (7, 8). Manelfe et al (6) found in a large series of embolized meningiomas that the low-density areas seen on postembolization CT corresponded histopathologically to necrotic tissue in only one third of cases. No necrotic tissue was found on microscopic examination in the remaining two thirds of the cases with hypodense areas on CT. Teasdale et al (9) reported necrotic changes in eight of 17 embolized meningiomas examined by CT and histopathology.

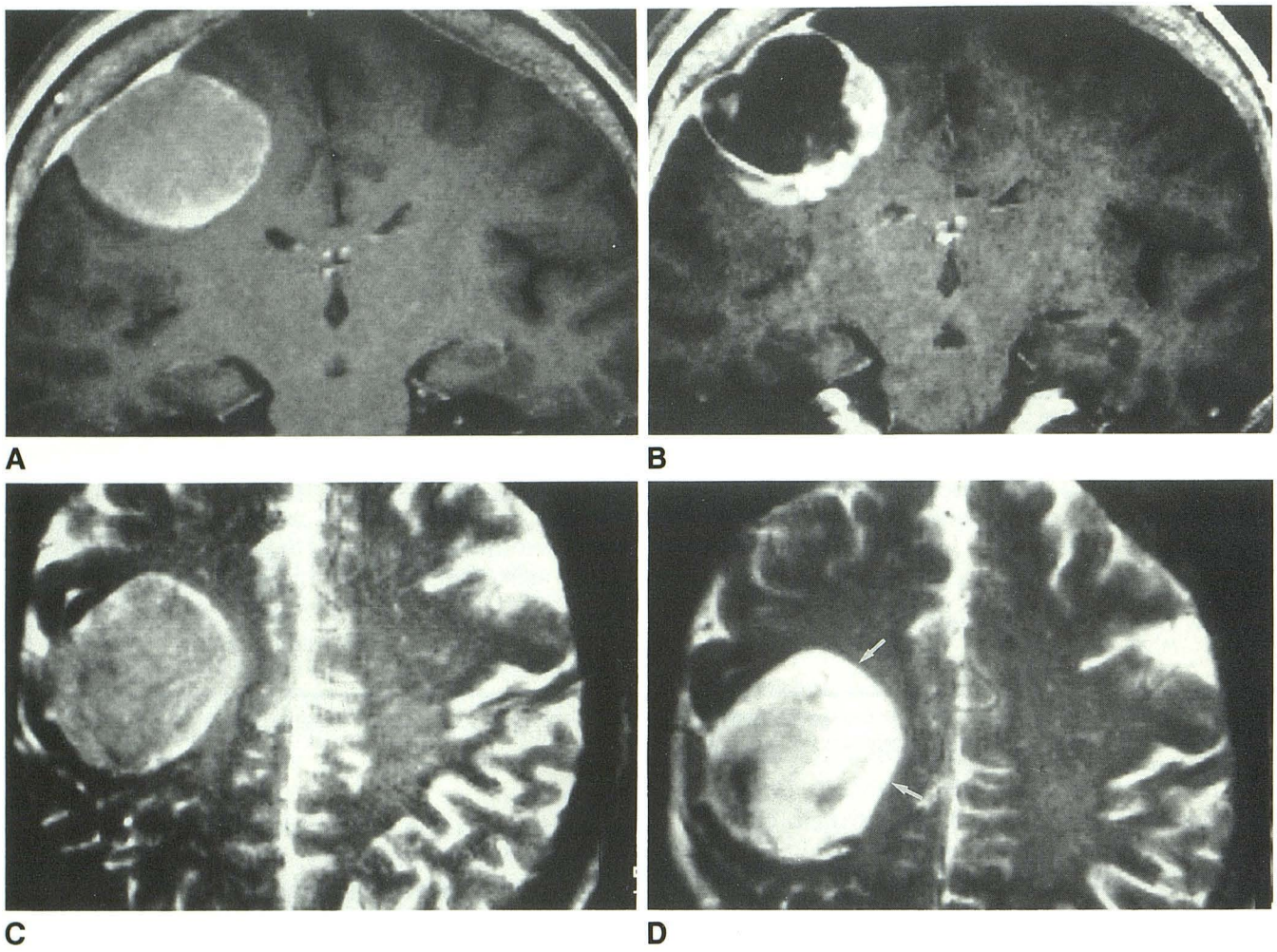

Fig. 4. Microembolization of convexity meningioma with 50- to 150-μm PVA particles (Table 2, case 2).

A, Contrast-enhanced coronal SE 500/20/2 MR image showing well-demarcated homogeneously enhanced tumor with a distinct dural thickening.

B, Subtotal devascularization of tumor after embolization. Note the shifting of the interhemispheric fissure and compression of the interhemispheric subarachnoid space by tumor swelling.

C and *D*, T2-weighted images (2450/32/2) before and after embolization, showing a distinct signal increase indicating liquefaction (*arrows*, confirmed histopathologically).

A recently published study by Jungreis (17) describes a significant reduction of intraoperative blood loss and scattered, histopathologically confirmed areas of necrosis after ethanol embolization via the cavernous carotid artery for petroclival meningiomas. However, the overall efficacy of this endovascular treatment cannot be evaluated from an imaging perspective, since no postembolization MR or CT studies are presented.

The present study analyzes postembolization changes of tumor enhancement and volume on CT and MR after using two different PVA particle sizes as embolic agent. Based on the postembolization MR scans, intraoperative ultrasound-guided biopsies were taken from different parts of the tumor or the adjacent dura. The operation was routinely performed within 1 week after the endovascular treatment. However, MR images obtained a few hours after embolization showed the beginning of tumor infarction, suggesting the feasibility of earlier surgery, as has already been reported (3). Nonenhancing tumor tissue corresponded to necrotic parts confirmed histologically. This fact supports the ability of Gd-DTPA-enhanced MR to define necrotic tumor mass after embolization. However, the ability of MR to detect small necrotic changes was limited, since a few necrotic or definitely necrobiotically changed tumor cells were found within some vital enhancing areas. The proton MR spectra of these tumor

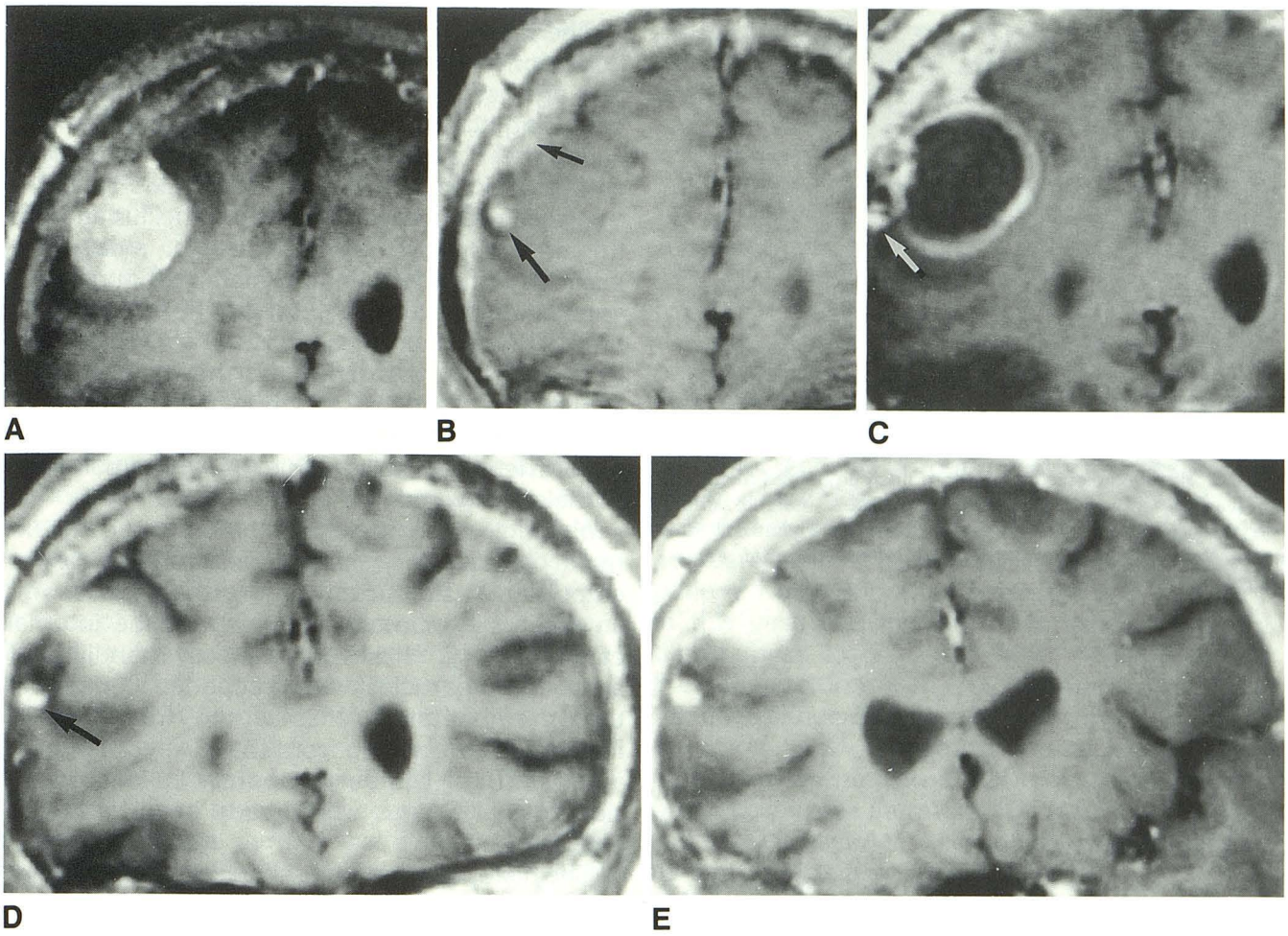

Fig. 5. Microembolization of convexity meningioma with 50- to 150-μm PVA particles (Table 2, case 13).

A, Contrast-enhanced coronal 3-mm SE 500/20/2 MR image showing homogeneous tumor enhancement.

B, Control MR image 4 hours after embolization revealing extensive devascularization. Note small, still well-vascularized areas adjacent to the dura (*arrows*).

C, Control MR images obtained 4 days after the endovascular occlusion show an enhancing tumor rim. Extensive central devascularized necrotic tissue.

D, MR performed 3 months after embolization shows a significant decrease of total tumor volume and reduction of the mass effect, leading to better demarcation of the adjacent sulcus. Persistant enhancing tumor nodule unchanged (*arrow*).

E, Control MR after 9 months showing no tumor regrowth.

parts revealed lactate as a sign of anerobic metabolism. In embolized nonenhancing tumor tissue accumulation of lactate or aliphatic lipid compounds was consistent with tumor hypoxemia and subsequent infarction.

The greater macroscopic devascularization and higher rate of tumor necrosis following the application of small rather than large PVA particles was the result of the extremely distal precapillary site of the embolization. This leads to a significant reduction of intraoperative blood loss. The tumor tissue, as well as the tumor-supplying dural feeders, were partly occluded with small PVA particles. This could be confirmed histopathologically in the Tibor Pap-stained specimens. Using larger embolic material, proximal embolization of the tumor-feeding vessels occurred. This may lead to a fairly good result insofar as the postembolization angiogram is concerned (6, 8). However, the tumor can still remain well vascularized, as seen on the contrast-enhanced MR images in most of our cases. Unwanted proximal arterial occlusion by large particles is generally responsible for the immediate revascularization of the tumor mass by collateral meningeal vessels bypassing the site of occlusion. This may be avoided by using 50- to 150-μm particles, since tumor capillaries have a diameter of 20 to 30 μm (18–20).

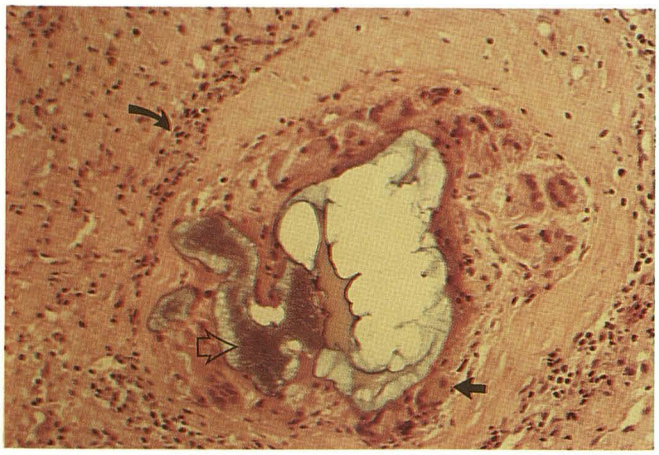

Fig. 6. Histopathologic findings in a microembolized meningioma (van Gieson staining). Intravascular embolic material (small PVA particles, 50- to 150-μm, *open arrows*) and giant cell formation (*short arrow*). Note intratumoral accumulation of granulocytes (*curved arrow*).

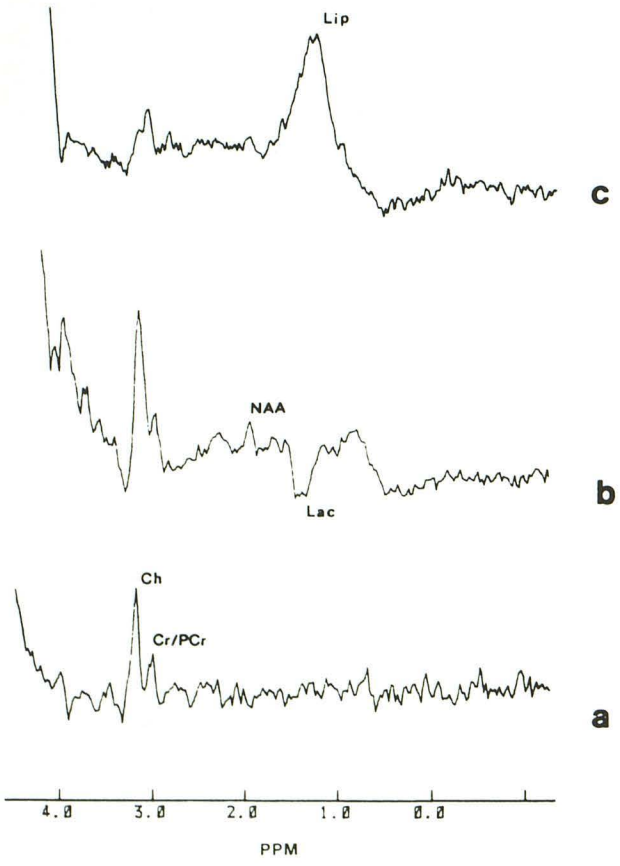

Fig. 7. Spectral pattern (1777/135/256) of an embolized meningioma (Table 2, case 9) before embolization (*a*), 4 hours after endovascular treatment (*b*), and 4 days later (*c*). Note antiphasic lactate peak at 1.3 ppm in *b* masked by broad lipid peak in *c*. *Cho* = choline; Cr/PCr = creatine/phosphocreatine, *NAA* = *N*-acetylaspartate; *Lac* = lactate; *Lip* = lipid.

The irregular surface of PVA has a high coefficient of friction, which permits the particles to rest against the vessel wall without completely occluding it. This irregular partial occlusion may cause the blood flow to stagnate, producing a combination of PVA and blood clot that will eventually recanalize with reendothelialization of the nonabsorbed PVA. The use of small PVA particles diminishes this problem, although their application will naturally prolong the embolization time. The injection must be given slowly in order to avoid dangerous reflux. Further technical advantages such as the reduction of particle aggregation in the microcatheter and reduced intraluminal friction are also achieved with small PVA particles delivered in a dilute solution.

Meningiomas supplied by branches of the external carotid artery alone benefit from the microembolization significantly more than those with an additional or major supply from the internal carotid artery. However, the easy handling of small PVA particles with fractioned centripetal tumor embolization enables the treatment of feeding vessels arising from the ICA, eg, tentorial artery, with or without temporary balloon occlusion of the ICA, as has already been described (10, 21).

The histopathologic classification of the meningiomas did not relate to the degree of necrosis after tumor embolization. So far as the angiomatous meningiomas are concerned, no definite conclusions can be drawn, inasmuch as only one such case was embolized in our present study.

Using small PVA particles, superselective catheter placement has to be employed to avoid scalp necrosis and the occlusion of vasa nervorum. This may be even more important when embolizing tumor vessels arising from the meningohypophyseal branches. Dangerous anastomoses with the vertebral artery at the C1/C2 level were seen during embolization in two cases of meningiomas of the posterior fossa, mainly supplied by branches of the occipital artery. In such circumstances, the microcatheter has to be placed quite close to the tumor nidus to avoid a reflux of embolic agent into the vertebral artery through the anastomosis. In none of the patients given endovascular treatment with 50- to 150-μm PVA particles was occlusion of normal brain vessels or intratumoral bleeding detected. However, in case 8 (group 1), a severe complication occurred 4 hours after the endovascular treatment of a frontal convexity meningioma with 150- to 300-μm PVA particles. Aphasia and hemiparesis devel-

oped because of intratumoral bleeding clearly demarcated on the CT scan. The control angiogram at the end of the embolization revealed an arteriovenous shunt not clearly depicted on the angiograms before the embolization. The cause of the bleeding could not be ascertained. The initial neurologic deficits related to the bleeding disappeared completely after operation. Such bleeding has been reported by other authors (22). Frequent control angiography should be carried out during the endovascular procedure to detect the opening of an arteriovenous shunt. We also think that fast embolization should be avoided in order to let the regional blood circulation adapt to the changing conditions during the treatment.

One major disadvantage of meningioma embolization in our series with small PVA particles was the distinct volume increase of the tumor in three patients (Table 2, cases 2, 5, 8) and an increase of perifocal edema in one patient. However, in two of these cases, no steroids had been administered before the embolization. Case 2 presented transient hemiparesis after the treatment, but recovered 2 days after operation. MR volumetric measurements revealed a 23% increase of the whole-tumor mass with compression of the adjacent precentral gyrus. Case 8 showed a 10% increase of whole-tumor volume, although only 5% tumor necrosis was seen after embolization. This patient, however, had not received sufficient steroid medication (4 mg, 3 times a day) before the endovascular treatment. The protective effect of steroids in endovascular treatment of meningiomas is suggested by our study, since in our three cases without sufficient steroid therapy, postembolization volume increase of the tumor was significant. However, this limited number of cases does not allow us to draw final conclusions.

Unlike experimental findings showing no significant inflammatory reaction to PVA foam depositions in the cerebral cortex of the rat (23), we frequently found extensive intravascular giant cell formation and, in some patients, a perivascular granulocytic infiltration, probably induced by the intravascular aggregation of 50- to 150-μm PVA particles. This observation is in accordance with a recently published follow-up study by Germano et al on arteriovenous malformations embolized with PVA particles (24). Such findings may be present even after steroid medication, as seen in most of our embolized tumors.

In two patients with convexity meningiomas referred to the hospital with nonspecific symptoms, no surgery followed the embolization. The one patient (Table 1, case 10) noticed a subjective improvement of her symptoms following the endovascular treatment with 150- to 300-μm PVA particles and refused surgery. Postembolization long-term control MR are not available yet. The other patient (Table 2, case 13) recovered completely from her neurologic deficits (hemiparesis, seizures, memory disturbance) after embolization with 50- to 150-μm PVA particles. The contrast-enhanced MR images obtained a few hours after the embolization (Fig. 5B) revealed large devascularized tumor parts. The appearance of a prominent rim enhancement on the fourth postembolization day (Fig. 5C) suggested revascularization of tumor capsule through pial branches of the cerebral arteries. Considering the clinical improvement and extensive tumor necrosis, the neurosurgeons refrained from operating on the patient. Follow-up after 3 months and 9 months in this case showed a further shrinking of the tumor mass to 36% and 30%, respectively, of the initial volume with a significant resorption of the central necrotic parts of the tumor. As far as we know, such a natural course of subtotally embolized meningioma has not been reported previously in the literature. Further investigations will be necessary to rule out any regrowth such as that reported by Koike et al (25).

We conclude that preoperative extended microembolization of intracranial meningiomas with 50- to 150-μm PVA particles induces a higher degree of effective tumor devascularization and necrosis than can be obtained with larger particles. No additional risks are associated with this technique. However, to avoid necrosis of the scalp or occlusion of vasa nervorum a superselective catheter placement is necessary. Besides that, a sufficient steroid medication before the endovascular treatment is recommended to prevent tumor swelling. This modified technique improves the tumor surgery, especially with lesions of the skull base. Furthermore, modified microembolization may be the only therapeutic intervention for those meningiomas that show a subtotal necrosis after the procedure, or in older or high-risk patients.

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