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BACKGROUND AND PURPOSE: **Preoperative embolization expands the spectrum of meningioma that can be operated on safely. Our goal was to achieve the distalmost loading of the vascular bed and confluent tumor necrosis with a fibrin glue preparation in the preoperative embolization of meningiomas.**

METHODS: **Between 1992 and 1997, 80 patients with a meningioma had diagnostic angiography with a standard transfemoral Seldinger technique, performed with a 6F guiding catheter and digital subtraction angiography. Preoperative embolization was carried out in the same session with an additional microcatheter system. Fibrin glue was the only component used. In all cases, CT was performed immediately after embolization; in nine patients, MR imaging was also performed.**

RESULTS: **Angiography verified the elimination of tumor blush in all patients. The high-density areas seen on postembolization CT scans, caused by the fibrin glue dispersed in the embolized supply area, were found to be necrotic at surgery and were easily removed by suction. Two (2.5%) of the 80 patients had complications associated with embolization that resulted in neurologic deficits.**

CONCLUSION: **The most effective preoperative embolization of tumors requires a distalmost loading of the vascular bed. Fibrin glue, which is easy to use and safe to handle, causes confluent tumor necrosis within the injected vascular territory.**

Although there are conflicting opinions concerning the value of preoperative embolization of meningiomas among neurosurgeons (1), this is now a standard procedure for reducing blood loss during surgery. The efficacy of embolization depends on achieving superselective catheterization of the supplying vessels and a distalmost loading of the vascular bed with embolic material. The embolization agent most often administrated is 150- to 300-μm polyvinyl alcohol (PVA) particles (2), but embolization with other materials, such as gelatin sponges, lyophilized dura, *n*-butyl cyanoacrylate, Silastic spheres (Dow Corning Corp, Midland, MI), and liquid material, has been described (3–5). While angiography often shows complete devascularization after embolization with these materials, contrast-enhanced MR tomography (2) and intraoperative and histologic findings generally have not confirmed tumor necrosis (3–5). Therefore, the embolization technique has been improved by the use of smaller particles (50–150 μm), which has led to more distal capillary embolization. Although this method does induce necrosis, it has the disadvantage of being more time-consuming, since injection of the 150- to 300-μm particles has to be quite slow to avoid dangerous reflux (6).

To achieve the distalmost loading of the vascular bed without the disadvantages associated with the use of PVA particles, we used a fibrin glue preparation (Tissucol Duo S, Immuno, Heidelberg) (7). This type of adhesive has been in standard use in various surgical specialties for tissue sealing and for obtaining hemostasis. The risk of transmission of infection is kept low, since the donors are regularly tested for alanine aminotransferase and for markers of viral infection. Allergic reactions are rare but may occur (8). Recently, a subgroup of patients has been identified in whom aprotinin-specific antibodies developed after topical aprotinin application (9). We report the radiologic, clinical, and histopathologic findings in a series of 80 patients in whom a fibrin glue preparation was used for the preoperative embolization of intracranial meningiomas.

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TABLE: Results of fibrin glue embolization in 80 patients with intracranial meningioma

Case	Age (y)/Sex	Tumor Location	Embolized Vessel	Complications
1	20/F	Convexity	MMA	Headache, vomiting
2	26/M	Convexity	MMA	· · ·
3	28/F	Convexity	MMA	· · ·
4	30/F	Convexity	MMA	· · ·
5	35/F	Convexity	APA, TOA	Headache, vomiting
6	38/F	Convexity	MMA	· · ·
7	40/F	Convexity	MMA	· · ·
8	41/M	Convexity	MMA	Nerve V palsy
9	43/M	Convexity	MMA	· · ·
10	43/F	Convexity	MMA	· · ·
11	44/M	Convexity	MMA	· · ·
12	47/F	Convexity	MMA	· · ·
13	50/M	Convexity	MMA	· · ·
14	50/F	Convexity	MMA	· · ·
15	51/F	Convexity	MMA	· · ·
16	52/F	Convexity	MMA	· · ·
17	53/F	Convexity	MMA	· · ·
18	54/M	Convexity	MMA, STA	· · ·
19	55/M	Convexity	MMA	· · ·
20	55/F	Convexity	MMA	· · ·
21	55/F	Convexity	MMA	· · ·
22	55/M	Convexity	MMA	· · ·
23	55/F	Convexity	MMA	· · ·
24	56/M	Convexity	EOA	· · ·
25	59/F	Convexity	MMA	· · ·
26	60/F	Convexity	AMA	· · ·
27	60/F	Convexity	MMA	· · ·
28	60/F	Convexity	MMA	· · ·
29	61/F	Convexity	MMA, EDA	· · ·
30	65/M	Convexity	MMA, STA	· · ·
31	68/M	Convexity	MMA	· · ·
32	70/F	Convexity	MMA	· · ·
33	70/M	Convexity	MMA	· · ·
34	71/F	Convexity	MMA	· · ·
35	71/F	Convexity	MMA	· · ·
36	74/F	Convexity	MMA	· · ·
37	74/F	Convexity	MMA	· · ·
38	78/F	Convexity	MMA	· · ·
39	12/M	Sphenoid wing	MMA	· · ·
40	24/F	Sphenoid wing	MaxA	· · ·
41	40/F	Sphenoid wing	MMA	· · ·
42	40/F	Sphenoid wing	MMA	· · ·
43	44/F	Sphenoid wing	MMA, MA	· · ·
44	45/F	Sphenoid wing	MMA	· · ·
45	45/F	Sphenoid wing	MMA	· · ·
46	45/F	Sphenoid wing	MMA	· · ·
47	46/M	Sphenoid wing	MMA	· · ·
48	52/F	Sphenoid wing	MaxA	· · ·
49	54/F	Sphenoid wing	MMA, APA	· · ·
50	55/F	Sphenoid wing	MMA	· · ·
51	62/F	Sphenoid wing	MMA	· · ·
52	64/M	Sphenoid wing	MMA	· · ·
53	68/M	Sphenoid wing	MMA	· · ·
54	69/F	Sphenoid wing	MMA	· · ·
55	70/F	Sphenoid wing	MMA	· · ·
56	70/M	Sphenoid wing	MMA	· · ·
57	72/M	Sphenoid wing	MMA	· · ·
58	56/F	Middle cerebral fossa	MMA	· · ·
59	33/F	Tentorium	MMA	· · ·
60	71/F	Tentorium	MMA, EOA	· · ·
61	47/M	Falx cerebri	MMA	· · ·
62	49/F	Falx cerebri	MMA	· · ·
63	51/M	Falx cerebri	MMA	· · ·
64	54/M	Falx cerebri	MMA	Urticaria

Table continued

Case	Age (y)/Sex	Tumor Location	Embolized Vessel	Complications
65	83/F	Falx cerebri	STA	· · ·
66	38/F	Cerebellopontine	MMA, EOA	· · ·
67	48/M	Cerebellopontine	APA	· · ·
68	60/F	Cerebellopontine	AMA, APA	· · ·
69	37/F	Posterior cranial fossa	MMA	· · ·
70	46/M	Posterior cranial fossa	MMA, APA	Headache
71	50/F	Posterior cranial fossa	MMA	· · ·
72	59/M	Posterior cranial fossa	AMA	· · ·
73	48/F	Clivus	MMA, APA	· · ·
74	52/F	Clivus	APA	· · ·
75	74/M	Clivus	APA	· · ·
76	38/F	Petroclivus	MMA	Incomplete facial nerve paresis
77	42/F	Petroclivus	MMA	Headache
78	64/F	Petroclivus	APA	· · ·
79	74/F	Petroclivus	MMA, APA	· · ·
80	62/M	Foramen magnum	APA	· · ·

Note.—MMA indicates middle meningeal artery; APA, ascending pharyngeal artery; TOA, temporal occipital artery; STA, superficial temporal artery; EOA, external occipital artery; MA, maxillary artery; AMA, accessory meningeal artery.

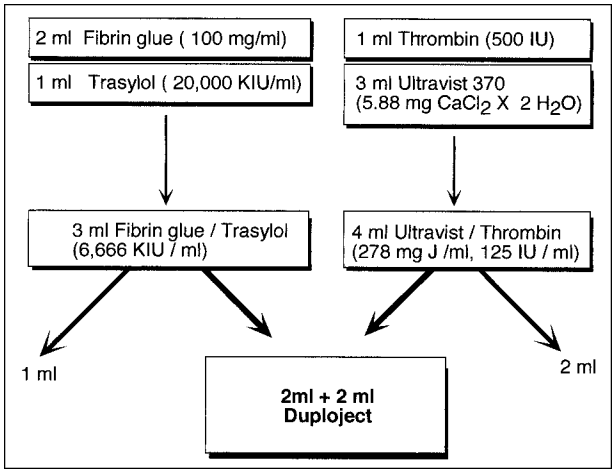

FIG 1. Preparation formula for the fibrin glue mixture. Final concentrations were as follows: 139 mg J, 62.5 IU thrombin, 33.25 mg fibrin glue, and 3.333 KIE Trasylol/mL.

Methods

Preoperative embolization with fibrin glue was undertaken in 80 patients with a meningioma. Fifty-four patients were female and 26 were male; mean age was 53 years (range, 12 to 83 years) (Table).

The patients presented with a wide spectrum of neurologic signs, with focal deficits and seizures being the most common. The patients underwent CT and/or MR imaging, whereupon a diagnosis of meningioma was presumed.

In preparation for preoperative embolization, we followed the basic ideas of Richling (10) and others (11, 12) to formulate a mixture of components (7) with the following criteria in mind: 1) could be continuously injected through a 165-cm-long microcatheter at 37°C, 2) would not precipitate too early inside the catheter or proximal vessels, 3) would be radiopaque in order to control injection, and 4) would produce a stable occlusion for at least 3 to 4 days.

Our tests resulted in the formula shown in Figure 1. We used a mixture of 2 mL human fibrin protein (Tissucol Duo S, Immuno, Heidelberg) (100 mg/mL) and, for an antifibrinolytic agent, 1 mL aprotinin (Trasylol, Bayer, Leverkusen) (20,000 KIE/mL). Two milliliters of this solution was put into half a double syringe (Duploject, Immuno, Heidelberg), of which the other half contained 2 mL of a mixture made up of 1 mL thrombin (500 IE/mL) and 3 mL Ultravist (Schering, Berlin) (370 mg J/mL, 5.88 mg $CaCl_2 \times 2\ H_2O$/mL). The mixture of Ultravist containing $CaCl_2$ was prepared by the pharmacy of the University Hospital Eppendorf. The total concentration in the double syringe of fibrin protein amounted to 33.25 mg/mL. The concentrated human fibrinogen is activated by the addition of human thrombin and calcium chloride. The antifibrinolytic agent aprotinin (bovine) is used to keep the mixture in solution. By using the double syringe, the material can be continuously injected through a Tracker 18 microcatheter (Target Therapeutics, Fremont, CA).

All 80 patients had diagnostic angiography with the standard transfemoral Seldinger technique, including a 6F guiding catheter and digital subtraction angiography. The catheter was continuously flushed with a solution of 5000 IU heparin/500 mL sodium chloride. Preoperative embolization was carried out in the same session with an additional Tracker 18 microcatheter system. The Tracker 18 catheter was introduced into the main feeding vessels, which, in most cases, originated from the external carotid artery. There was no provocative testing prior to embolization, as the risk of complications after embolization within the external carotid artery is very low.

At the beginning of the embolization procedure, the material was slightly visible under fluoroscopy if injected slowly. With intermittent small-bolus injections, the continuous reduction of flow into the tumor was easily monitored (Fig 2). Postembolization angiograms were obtained with the therapeutic catheter in place and again using the diagnostic catheter in the proximal part of the external carotid artery. The material was mixed freshly just before injection.

In all patients, fibrin glue was the only component used, and even for large tumors we rarely needed more than 6 mL. Surgery was performed in all cases within 6 days, except in one patient (case 73).

All patients had CT immediately after embolization to delineate the contrast-enhanced embolized areas within the tumor (Fig 3). In nine patients, MR imaging was also performed.

Results

The results of preoperative embolization with fibrin glue in the 80 patients with a meningioma are

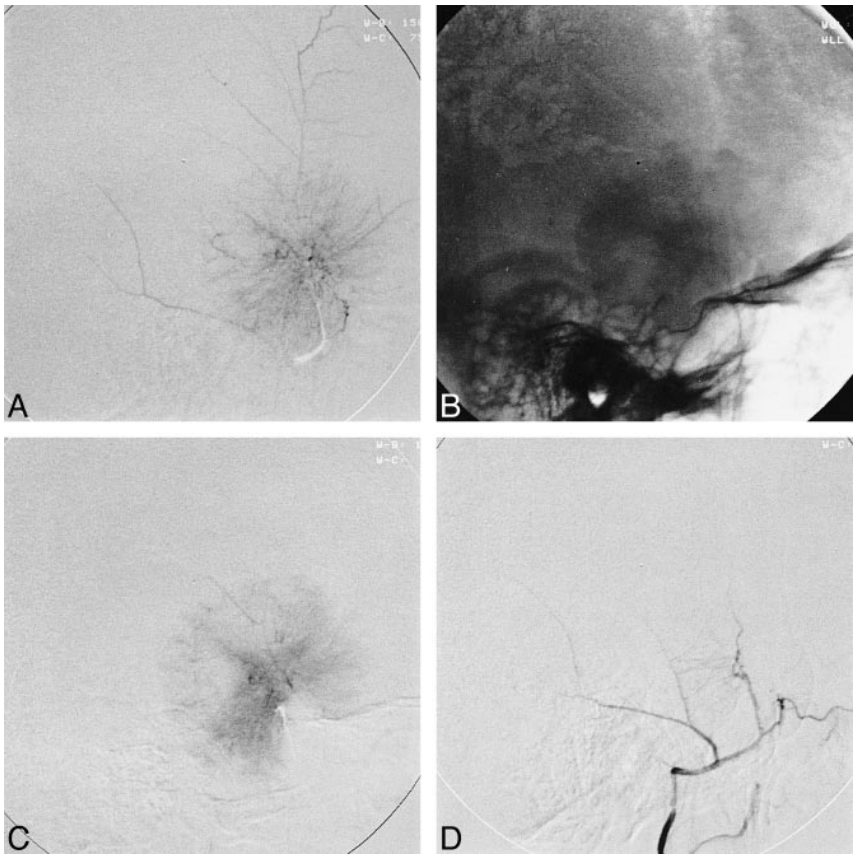

FIG 2. Case 42: 40-year-old woman with sphenoid wing meningioma.

A and *B,* Lateral views of vascular arterial tumor supply show the main feeding artery to be the middle meningeal artery on the right side.

C and *D,* Capillary phase (lateral view) shows progression of embolization with fibrin glue.

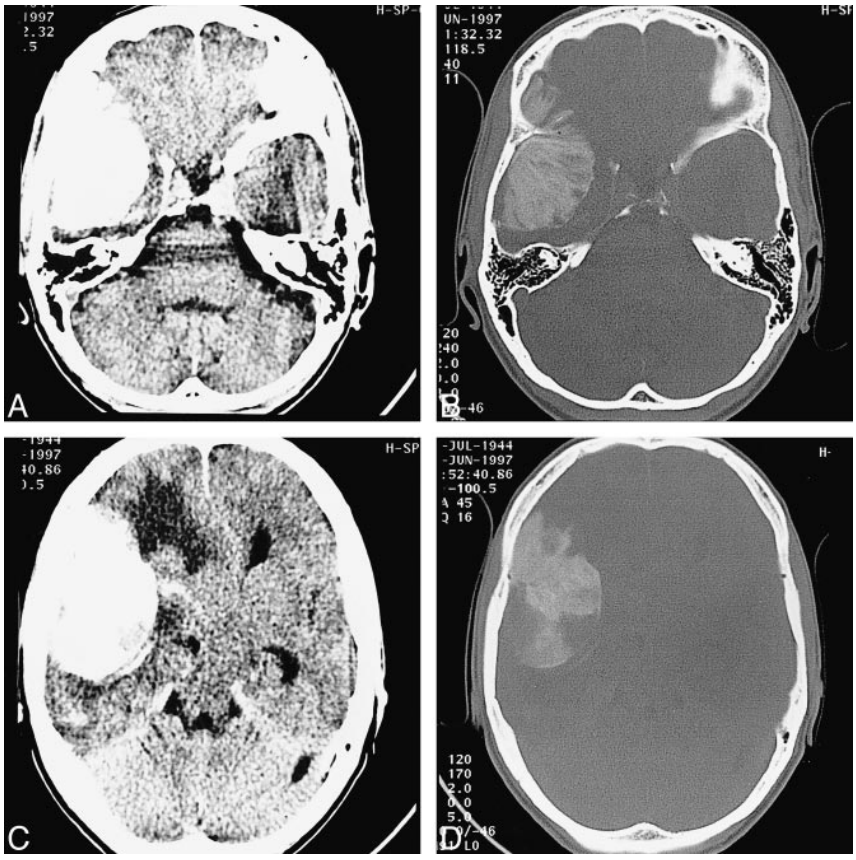

FIG 3. *A–D,* CT scans of same patient as in Figure 2 after embolization of the tumor core fed by the middle meningeal artery. Considerable hyperdensity, caused by fibrin glue, is dispersed homogeneously in this supply area. Less hyperdensity, from contrast medium alone, is seen within the nonembolized areas.

given in the Table. In 65 patients the tumor was located supratentorially, and in 15 it was found infratentorially. Among the patients with a supratentorial tumor, 38 had a convexity meningioma larger than 5 cm in diameter. Nineteen patients had a lateral sphenoid wing meningioma predominantly fed by the middle meningeal artery; five were located in the parasagittal and/or falx region, partially or completely occluding the superior sagittal sinus. In one case, the meningioma was located in the middle cranial fossa, in two other patients, at the tentorium.

The infratentorial meningiomas were found at the clivus/foramen magnum (eight cases), at the cerebellopontine angle (three cases), and at the posterior cranial fossa (four cases). Among the supratentorial meningiomas, the main arterial feeder was the middle meningeal artery. In some cases there was also a contribution from the ascending pharyngeal, temporal occipital, superficial temporal, external occipital, maxillary, or accessory meningeal artery. Among the infratentorial tumors, the main feeding vessels were the middle meningeal and the ascending pharyngeal arteries.

Most of the time spent in the embolization procedure was for superselective catheterization; the injection of fibrin glue itself was completed within a few minutes, including the postembolization control studies.

The effects of embolization on the surgical procedure were mixed. In general, the areas that showed contrast enhancement on the postembolization CT scans (Fig 3), indicating that fibrin glue had entered the parenchyma of the tumor, were found to be necrotic and could be removed by suction, thus achieving rapid internal decompression. Because such decompression facilitates dissection of the meningioma away from the frequently edematous brain, this is a beneficial effect. Especially in otherwise very tough tumors, this is an alternative to the cutting loop, which may leave bleeding surfaces behind, sometimes resulting in substantial blood loss. This necrotizing effect, however, is unequivocally beneficial only so long as the necrotic areas are in the central part of the meningioma. In those cases in which large areas of the tumor became necrotic and whole sectors of the tumor were affected, involving the surface of the tumor, the dissection plane between the meningioma and the surrounding brain became fuzzy, making removal more difficult, theoretically increasing the possibility of compromising the intact perilesional tissues. This, however, was the exception, occurring in only four cases, without harm to the patient.

At histologic examination, which was performed for every surgically removed tumor, the first manifestation of necrosis was apparent the day after embolization (Fig 4). Because the fibrin glue cannot be seen histologically, its presence may lead to diagnostic confusion in terms of the grading of meningiomas. Necrosis is considered a hallmark of anaplasia, and as such justifies the allocation of

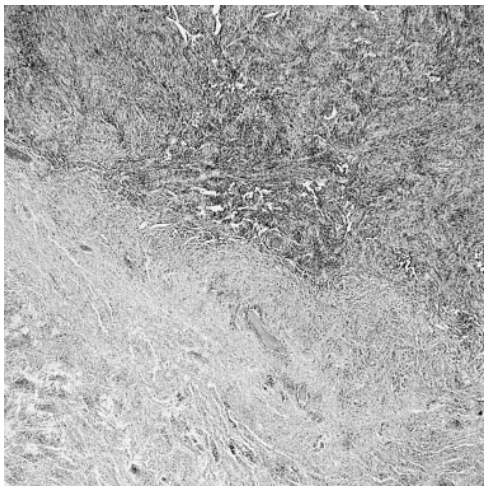

FIG 4. Histologic specimen shows the clear-cut borderline between necrotic meningeal (*lower right*) and vital arachnoidal (*upper left*) supply areas after embolization (hematoxylin-eosin, original magnification ×40).

WHO grade III, which then also implies that radiotherapy or chemotherapy is to be considered. In this series, some clues have been worked out, such as the synchronous timing of necrosis in truly anaplastic tumors (13). In addition, the Ki67 labeling index adjacent to embolic necrosis is higher than in still vital tissue, also a difference from regular anaplastic meningiomas (14).

A slight headache during the embolization procedure was experienced by all patients; a splitting headache and nausea, probably due to swelling and requiring symptomatic treatment, were experienced by four patients. All patients were treated with dexamethasone, 4 mg every 8 hours.

The preexisting clinical symptoms were not affected by the embolization in any case. Two patients had complications associated with the procedure that resulted in neurologic deficits (Table, cases 8 and 76). One of them (case 76), a patient with a petroclival meningioma, had an incomplete paresis of the facial nerve immediately after the procedure; however, recovery was complete 1 year after embolization and surgery. The other patient (case 8), a 41-year-old man with a parietal meningioma fed primarily by the middle meningeal artery, experienced hypoesthesia in the sensory territory of the third trigeminal nerve immediately after embolization, which was persistent 1 year later.

Among the 15 infratentorial tumors, eight were found at the clivus. In one of these patients (case 46), presenting with cephalgia, dizziness, and a tingling sensation in the second and third trigeminal nerves on the right side, the tumor displaced the pons and compressed the fourth ventricle (Figs 5 and 6). The main feeder was the right ascending pharyngeal artery, but the left ascending pharyngeal artery and the middle meningeal artery were also involved. Four days after embolization, a contrast-enhanced T1-weighted MR image showed ex-

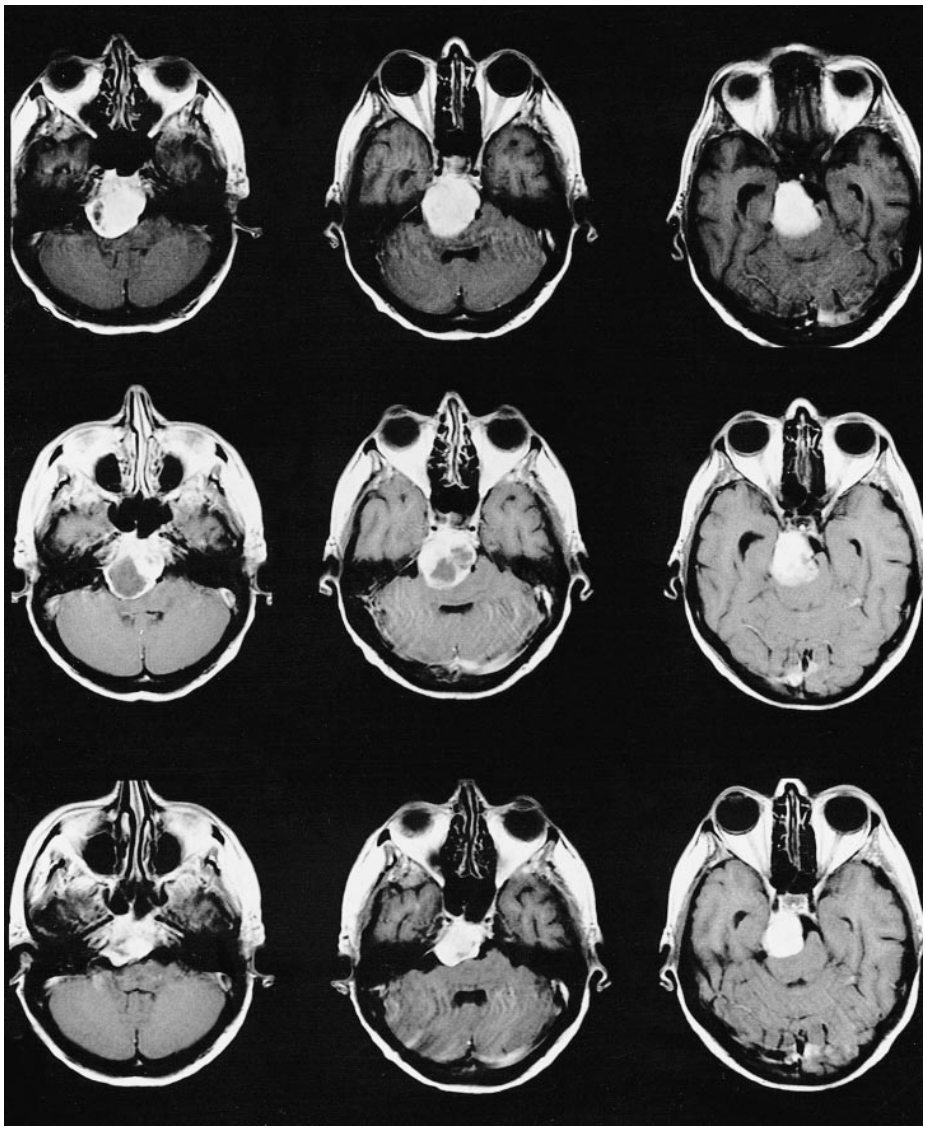

FIG 5. T1-weighted axial MR images of a clivus meningioma obtained after administration of contrast material.

Top row, Images obtained before embolization show the tumor displacing the pons and compressing the fourth ventricle on the right side.

Middle row, 4 days after embolization of the meningioma, extensive necrotic areas are visible, especially in the rostral and dorsal parts of the tumor.

Bottom row, 5 months later, tumor is significantly reduced in size. The necrotic areas are resorbed and the compression of the pons and the fourth ventricle is barely detectable.

tensive necrotic areas, especially in the rostral and dorsal parts of the tumor. Five months later, the MR images showed a significant reduction in the size of the meningioma. The necrotic areas were resorbed, and the compression of the pons and the fourth ventricle was hardly detectable. At surgery, 6 months after the embolization procedure, a benign transitional meningioma of the clivus was found. Interestingly, the surgeons reported an unusual, rubbery consistency of the tumor, impeding its removal (Personal communication, L. Cristante, Winnipeg, Canada).

Discussion

Recently, Wakhloo et al (6) reported a reduction in tumor enhancement in only two of 14 patients in whom contrast-enhanced MR images were obtained after treatment with 150- to 300-μm PVA particles. Similarly, Grand et al (15) reported a decrease in MR enhancement of only 2.5% after embolization in seven of 15 patients who had an 80% supply from the external carotid artery. The authors attributed this finding to spasm. Latchaw (1), supported by the results of Wakhloo et al (6), proposed that this discrepancy was probably due to the large size of the particles (150–300 μm), which caused the vasculature to be blocked more proximally. This phenomenon, which is probably due to premature clumping of the particles with proximal occlusion of the vessels, explains earlier published findings of complete devascularization without corroboration of CT and intraoperative findings (5). It

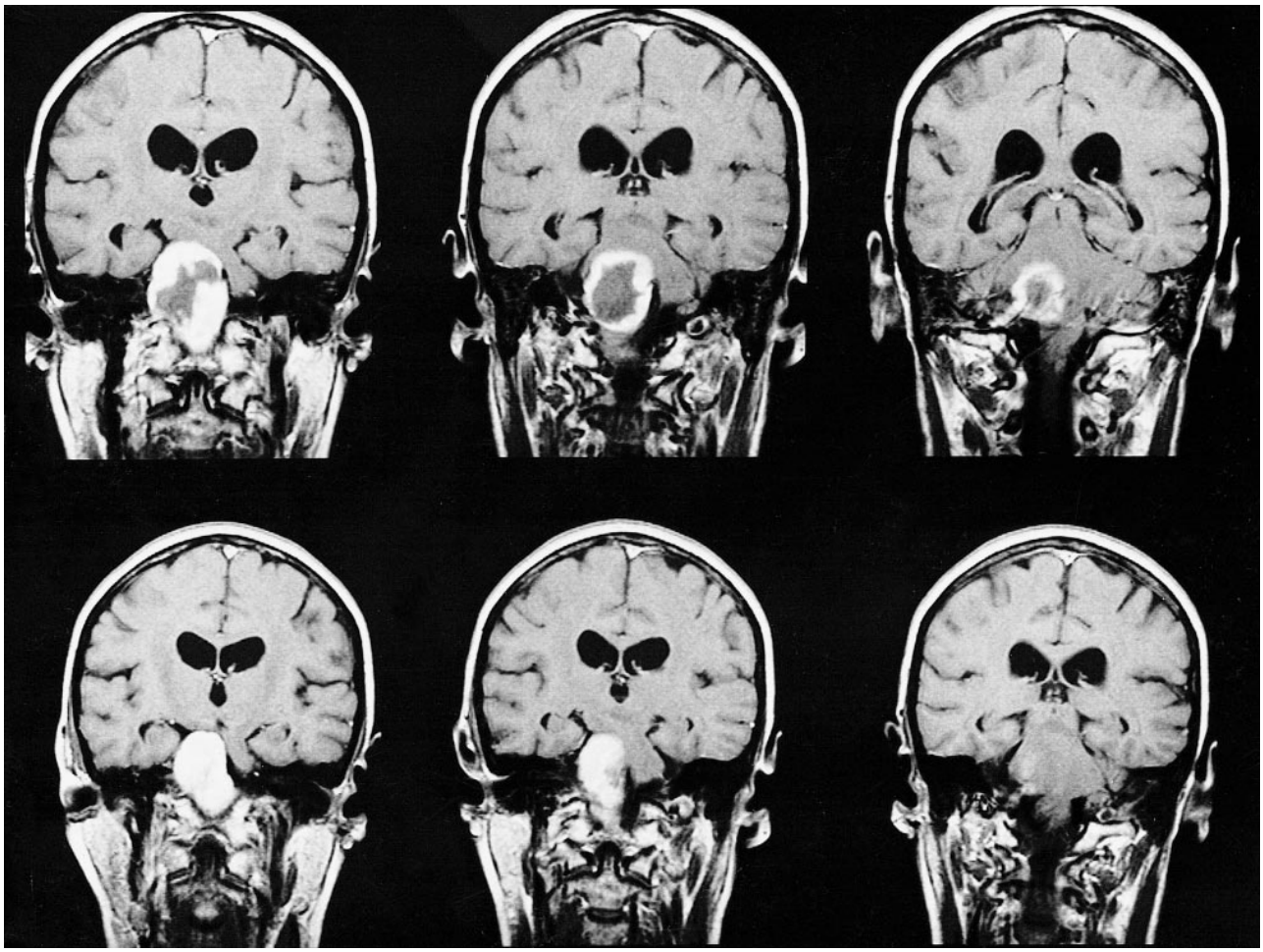

FIG 6. Coronal T1-weighted MR images of the same patient as in Figure 2 after administration of contrast material show reduction in tumor size.

Top row, 4 days after embolization, the meningioma shows extensive necrotic areas.

Bottom row, 5 months later, there is resorbtion of the necrotic areas and a reduction in tumor size.

also explains why neurosurgeons may perceive embolization as ineffective and consider its implementation with some reservation, especially since they can easily do a proximal vascular occlusion intraoperatively themselves.

After the methodology was refined further by using smaller (50- to 150-μm) PVA particles, significant tumor necrosis could be seen on MR images and confirmed histologically after surgery (6). The smaller PVA particles seem to cause a more distal precapillary embolization, greater devascularization, and a higher rate of tumor necrosis than the 150- to 300-μm PVA particles. This confirms the need for distalmost loading of the vessels for effective preoperative embolization. One major disadvantage of the smaller (50- to 150-μm) PVA particles is that it seems to be an extremely time-consuming procedure. In our experience, embolization time was 60 to 120 minutes for each feeding vessel, sometimes even as long as 150 minutes.

Employing a different approach, we used a fibrin glue (Tissucol) preparation (Fig 1) as an alternative to the time-consuming PVA particles. Since this mixture dissolves immediately in larger vessels, superselective catheterization of all accessible feeding vessels is crucial. This explains why most of the time spent embolizing with fibrin glue is used for superselective catheterization. The glue injection itself is completed within a few minutes (see Results), including the postembolization control studies. Owing to the radiopaque nature of the fibrin glue mixture, continuous monitoring during embolization is possible. This, together with a CT scan obtained immediately afterward showing the extent of loading of the peripheral vascular bed, can document the success of the procedure. Intraoperative findings and histologic work-up both demonstrate tumor necrosis in the embolized areas.

Complications related to the embolization and resulting in neurologic deficits did occur in two patients (cases 8 and 76, see Table). Hypoesthesia in the innervation area of the third trigeminal nerve is an unusual complication of embolization of the middle meningeal artery. In 1976, Lasjaunias and Theron (16) described small vessels originating from the accessory meningeal artery penetrating the foramen ovale at the ganglion trigeminale. This might have been the situation and the reason for

the complication in our patient. No patient experienced neurologic deterioration due to swelling after embolization. Minor complaints and aggravations caused by postembolic reaction of the perilesional brain were managed and contained by the obligatory steroid medication before endovascular and surgical treatment. Slight headache during the embolization was experienced by all patients; splitting headache and nausea requiring symptomatic treatment were experienced by four patients, and were probably due to the embolization of small branches of the meningeal artery associated with the tumor, which could not be avoided. Recanalization due to the intrinsic fibrinolysis was not found to affect the intraoperative results to any significant degree. On the contrary, the material induces tumor necrosis to such an extent that recanalization might play a role only in borderline zones with mixed meningeal and arachnoidal supply. Because our patients underwent surgery within 6 days of embolization, the risk of recanalization of the large feeding meningeal arteries and possible hemorrhage into the necrosis was unlikely. Another reason for early surgical intervention, if surgery is planned, had to do with the consistency of the tumor after embolization. For example, in one of our patients (case 73), a significant reduction in tumor size was achieved after embolization of a clivus meningioma. The patient underwent surgery 6 months later, after she had recovered from her neurologic deficits. At this time, however, the surgeons found the tumor hard to remove because of its rubbery, scarlike consistency.

Our study shows that superselective injection of fibrin glue into the vascular bed of a meningioma is a safe and effective method for reducing blood supply to the tumor. Nevertheless, despite its proved safety and feasibility, preoperative embolization will not become a general standard for meningioma therapy, as many lesions are easily devascularized by routine surgical techniques and will be handled in institutions without interventional neuroradiologic support. There seem to be some indications, however, as have emerged from our study of 80 patients, that preoperative embolization may become a standard component in certain situations. For example, embolization is especially useful in cases in which the blood supply will be reached only at the end of the operation unless extensive bone removal is performed as the initial step of the surgical procedure, such as for lesions at the dorsal aspect of the petrous bone, where the blood supply comes from the mastoid meningeal branches. Also, for elderly patients, the duration of surgery and extent of blood loss can be minimized by preoperative embolization. Finally, preoperative embolization is useful for patients with large sphenoid wing or tentorial meningiomas who, for personal reasons, refuse to consent to blood transfusions. Among our group of patients, we had one elderly patient with a large sphenoid wing meningioma who was refused treatment by several other institutions and who underwent surgery with only minimal blood loss 2 days after embolization.

Conclusion

Preoperative embolization with fibrin glue expands the spectrum of meningiomas that can be operated on safely. In our experience, although not derived from a randomized, prospective, controlled trial, intraoperative blood loss and operating time were reduced, which was of special benefit to the mostly elderly patients. Surgery performed within 1 week of embolization appears to be the optimal timing.

Acknowledgments

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