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Occlusion of Cerebral Arteriovenous Malformations with *N*-Butyl Cyano-acrylate Is Permanent

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PURPOSE: To verify the permanence of total occlusion of cerebral arteriovenous malformations after embolization with *N*-butyl cyano-acrylate and to evaluate the occlusion rate. **METHODS:** One hundred thirty-four patients were treated for cerebral arteriovenous malformations with *N*-butyl cyano-acrylate embolization after superselective catheterization. Those initially totally occluded have been followed with angiography. **RESULTS:** In 15 instances (11.2%) the arteriovenous malformations were totally occluded by embolization alone. Follow-up angiograms have been performed in 12 cases (80%) after 4 to 78 (mean, 27) months after embolization. In all instances the arteriovenous malformations have remained occluded, and there have been no clinical signs of activity. **CONCLUSION:** After total occlusion of a cerebral arteriovenous malformation with *N*-butyl cyano-acrylate, the lesion stays occluded, and the clinical course seems stable.

Index terms: Arteriovenous malformations, embolization; Interventional materials, embolic agents

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Several treatment options exist regarding cerebral arteriovenous malformations (AVMs) of the brain. One of these is transarterial embolization, which has now been used for more than 30 years (1), and materials and methods have developed considerably over time. One fundamental question when embolizing AVMs is whether the treatment is permanent or if the AVM will recanalize over time. This paper presents our experience in this respect regarding the embolic agent *N*-butyl cyano-acrylate.

Materials and Methods

From January 1, 1987, to December 31, 1992, 134 patients were treated at our institution with embolization for AVMs. No patient was treated outside this interval of time. The patients had mostly been deemed bad risks for surgery or not suitable for stereotactic radiosurgery because of AVM size or location or patient compliance at different neurosurgical clinics in the catchment area for interventional neuroradiology. Of these, 15 (11.2%) were totally occluded with embolization solely, without remaining angiographic signs of nidus or shunt. These patients make up the study group.

The method of embolization was initially as described by Kerber (2) in 1976: the flow-guided, calibrated-leak balloon technique. It was mainly used until 1989 and occasionally later. As the variable stiffness guide wire–guided microcatheter (3) (Tracker, Target Therapeutics, Fremont, Calif) became available, it was used more from 1987 to 1990, when it was gradually replaced by the very floppy, flow-guided pursil catheter (4) (Magic, Balt, France). Of the patients with totally occluded AVMs, *N*-Butyl cyano-acrylate (Histacryl Bleu, Melsungen AG, Germany) was used in all; polyvinyl alcohol was used as an adjunct in one. No other embolic materials were used.

The sizes of the AVMs have been measured on the films. Veins have not been included. Magnification and minification factors have always been taken in account. The AVMs have been approximated to ellipsoids and their volumes calculated using the formula:

$$V = d_1 \times d_2 \times d_3 \times \tfrac{1}{2},$$

where d_{1-3} are the three diameters of the AVM in centimeters, and V is the volume in milliliters (5).

Radiologic follow-up has been through cerebral angiograms performed usually at the referral hospital in the area where the patient lives. The angiograms have included all possible feeders to the area of the AVM. The criteria for total occlusion has been no remaining abnormal nidus vasculature or shunting and early-filling veins. All angiograms have been examined by us. Clinical follow-up was

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TABLE 1: Lesion diameters and volumes in the whole group, in the group not totally occluded, and in the group totally occluded with embolization alone

Lesion	Whole Group	Group Not Totally Occluded	Group Totally Embolized
Number	134	119	15
Diameter, mm	41.6 ± 15 (range, 10–120)	44.8 ± 15.3 (range, 15–120)	23.1 ± 10.4 (range, 10–40)
Volume, mL	26 ± 32 (range, 0.5–280)	28.4 ± 33.2 (range, 1–280)	7 ± 7 (range, 0.5–26)

Note.—Values are mean ± SD.

performed via notes in patient files, done when follow-up angiography was performed.

Results

The AVMs totally occluded differed in size from the whole group of AVMs referred for embolization. They were smaller (Table 1). The rate of total occlusion among AVMs of different sizes is presented in Table 2.

The 15 patients in whom embolization was the only treatment have been followed up with angiography in all instances except 3. One patient with a thalamic AVM that had bled and had given her a hemiparesis had an increased pareses after embolization; this patient has been negative about all contact. The second patient has a psychiatric disease preventing angiographic follow-up, and the third patient died from pancreatic cancer 2 years after treatment. That leaves 12 (80%) cases being controlled with angiography after 4 to 78 months after embolization (mean, 27 months). In no instance was there any sign of revascularization of AVM; that is, there was absence of abnormal nidus vasculature and absence of shunting. The clinical follow-up has been for a total of 44 patient years. During this period no patient has had any bleeding or other new symptoms referable to the AVM.

Table 3 shows numbers of patients in relation to catheters used for embolization and numbers

TABLE 2: Rate of total occlusion among AVMs using 25 and 30 mm, respectively, as size limit for small AVMs

Size Limit, mm	Total Number (%)	Number Occluded	Percentage Occluded
≤25	14 (10.4)	9	64.2
>25	120 (89.6)	6	5.0
≤30	26 (19.4)	11	42.3
>30	108 (80.6)	4	3.7

TABLE 3: All patients divided into groups according to catheter used for embolization, the occlusion rate achieved in each group, and the volumes of the AVMs

Technique	Total Number	Number Totally Occluded	Percentage Totally Occluded	Lesion Volume, mL, mean ± SD
Tracker and/or Kerber	64	11	17.2	27 ± 39
Magic	70	4	5.7	24 ± 22

Note.—The difference in proportions totally occluded is significant; $P < .02$.

and percentages of total occlusion in each group. There is an overlap between the groups, but no Magic catheter has been used in conjunction with any other type catheter in a patient with a totally occluded AVM.

Discussion

Since embolization became an alternative treatment to surgery for cerebral AVMs, there has been discussion of which therapeutic method is the safest. The "no treatment at all" option, especially for nonruptured AVMs, has its spokespeople (6). The decision to treat or not to treat depends heavily on knowledge of the natural history of this disease. Much has been written and bias in the studies discussed, but there is a rather uniform opinion that the cumulative yearly bleeding rate is around 3% and the yearly mortality from the disease is around 1% (7–9). Ondra et al (7) claim no difference in these risks regardless of presentation with hemorrhage or seizures. Brown et al (8) found approximately the same risk as cited above in his material of nonbleeders.

These studies show rather convincingly that the risk for complication because of an AVM does not depend on size or mode of presentation. Our policy has been to treat AVMs if angiography has shown appropriate paths to reach the nidus. We also considered the risk of hemorrhage to be increased if an intranidal aneurysm is present (10–12). Deruty et al found in their series that despite aneurysms in the area of the AVM, the malformation was the source of bleeding in all six studied instances (13). According to our experience, nidal aneurysms might hide in the nidus and not appear until superselective angiograms are performed or even after partial embolization. With improved catheters, it has been possible to reach the nidus in most cases referred to us. In about 10% it

has not been possible to reach a superselective position at which no functional parenchyma was involved. The 11.2% of the AVMs that have been successfully occluded by embolization alone is among the lower frequencies reported in the literature (14–17), but lesion sizes are not well reported. In our group of small AVMs (≤25 mm), the frequency of total occlusion is 64.2% (Table 2).

The permanence of embolization has been discussed and questioned (18). The cases reported by Rao et al (18) show that contrast in the cast has disappeared but that collaterals have developed to supply nonembolized nidus. The same phenomenon is stated by Fournier et al (19).

Our experience is that *N*-butyl cyanoacrylate is superior to polyvinyl alcohol regarding permanence. A recent paper from Nakstad et al shows interesting results with decreased recanalization rate after polyvinyl alcohol combined with platinum coils (20). This type of embolization demands Tracker or similar catheters, and in our experience it is not possible to achieve as distal catheterization with these catheters as with the Magic. Often one might therefore be dependent on the sump effect of the shunt to get the emboli to the right location. When the sump effect decreases, the risk for inadvertent embolization might increase. Development in this field is steadily ongoing, and the new hydrophilic coated Fastracker (Target) may well permit more distal catheterization. However, the possibly increased risk with use of guide wires intracranially, especially in vessels with high-flow angiopathy (21), remains with this technique.

If the rebleeding rate cited above (3%) were used on a group of 15 patients, statistically there should be a hemorrhage every second year. Because our average clinical follow-up time is 35.5 months, follow-up time is too short for definite conclusions regarding clinical cure in this material. However, the fact that none of our totally treated patients have had any sign of recanalization and no clinical sign of remaining AVM activity is a strong indicator that embolization with *N*-butyl cyano-acrylate is a permanent cure.

When splitting the material into different groups according to catheters used (Table 3), there seems to be an advantage to total occlusions when the Tracker- or Kerber-type balloon is used. If this difference is true, one reason might be the increased ratio of *N*-butyl cyanoacrylate to blood with the Tracker (caused by larger inner lumen) or flow control with the Kerber balloons. Embolization with these catheters might allow better penetration of the nidus.

Conclusion

Arteriovenous malformations of the brain occluded with *N*-butyl cyano-acrylate remain occluded, and the clinical course after treatment seems stable.

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