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Benefits of Embolization without Surgery for Cerebral Arteriovenous Malformations.

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Embolization may be the primary and exclusive treatment of cerebral arteriovenous malformations. To evaluate its benefits, a group of 27 patients was studied for the incidence of hemorrhages, headaches, seizures, and neurologic deficits, both before and after Silastic sphere embolization. Questionnaires to patients and physicians were used. Embolization was found to decrease the frequency of headaches in most patients with this symptom, but had no effect on the frequency of seizures when patients in whom medical therapy was altered were excluded nor on the progression or incidence of neurologic signs and symptoms. The effect of embolization on the incidence of recurrent hemorrhages is as yet undetermined.

Cerebral arteriovenous malformations are vascular hamartomas with arteriovenous shunts [1]. They occur more often in the brain than elsewhere in the body [2] and are the most common of all the vascular malformations of the central nervous system. Their incidence in the United States population has been estimated at 0.14% (280,000 people) [3]. They accounted for about 0.6% of the patients with nontraumatic subarachnoid hemorrhage in one study [4]. In addition to hemorrhage, which has been reported as the main complaint in 30%–76% of the patients [5, 6], many patients had headaches or seizures.

Most cerebral arteriovenous malformations probably enlarge progressively, although cases of regression or spontaneous disappearance have been reported [7]. Because of the potential devastating effects of an intracerebral hemorrhage, therapy is usually directed to surgical removal or embolization of the lesions. Embolization has been advocated also as an aid to surgery. While it is known that surgical removal of the lesions completely removes the risk of recurrent hemorrhages, the effects of embolization alone on the natural history of the lesions and their accompanying symptoms is largely unknown. We report a follow-up study of the incidence of hemorrhages, headaches, seizures, and neurologic deficits in a selected group of patients with cerebral arteriovenous malformations treated exclusively with carotid and vertebral embolization. The information was obtained from the patients and from their attending physicians.

Subjects and Methods

Patient Population

During a 7 year period, 63 patients with cerebral arteriovenous malformations were embolized. In 32 patients, surgery was considered feasible, but the malformations were located in parts of the brain where surgical control of the feeding arteries was thought to be difficult or the malformations were considered too large for successful removal without significant morbidity, thus necessitating presurgical embolization. All 32 patients subsequently underwent surgery.

In a second group of 31 patients, the malformations were considered too large to be successfully removed surgically or they were situated in critical areas of the brain where

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surgery was not considered possible. Embolization alone was carried out in this group to attempt to reduce the size of the malformations and their feeding arteries and draining veins. Correction of the altered hemodynamics of the cerebral circulation was also considered desirable. In both groups, the benefits of reducing the size of the feeding arteries or of the arteriovenous malformation itself were considered to outweigh the risk of embolization.

Follow-up

In the second group of 31 patients, the follow-up information was obtained from questionnaires mailed to the patients and their physicians. Responses were received from 23 (74%) of the 31 patients and from 11 (36%) of their physicians. The follow-up patient group was composed of the 27 patients who either responded themselves or whose physicians responded or both. Five of the physician responses regarded patients who did not respond themselves. In four cases, we received no response either from the patients or their physicians; in two of these patients, because of the large size of the shunts within the malformation, embolization was known to have been inadequate.

The patient questionnaire inquired about recurrence of hemorrhage, severity and intensity of headache, seizure activity, and neurologic changes. If more than one embolization procedure was carried out, patients were asked about their response to *each* procedure. The physician questionnaire requested the physician's evaluation of the patient's status and of the contribution that the embolization(s) had made to each patient's care.

Technique

Embolization was carried out via the percutaneous transfemoral approach using the Seldinger technique. Catheters were placed in the appropriate carotid or vertebral artery. Prior to embolization, 10 mg of Decadron were administered intravenously. Barium-impregnated Silastic emboli were injected, and their location in the brain was determined by frontal and lateral skull films. In 26 patients, 2 mm or smaller emboli were injected; in one patient, both 2 and 3 mm emboli were injected. The procedure was terminated after 60 emboli were injected, when emboli were seen to stray into normal arteries, or when neurologic complications occurred [8].

Both pre- and postembolization angiograms were obtained in all patients. The postembolization angiograms were studied to determine the hemodynamic benefits of embolization. Partial obliteration of the arteriovenous malformation (in no patient was the malformation completely obliterated), slowing of the circulation time through the malformation, stasis of contrast material within the feeding arteries to or draining veins from the malformation, and occlusion or decreased size of the feeding arteries to the malformation were all considered beneficial. A return to a normal circulation with elimination or decrease of an arterial steal was also considered desirable.

In each of 18 patients, 20 or more emboli were injected into the cerebral arteries. Beneficial results were obtained in all 18. In 13 patients, fewer than 20 emboli were injected; nine received 10–20 and four received fewer than 10. In the group of nine patients, examination of the postembolization angiograms demonstrated no change in the hemodynamics of the arteriovenous malformation in eight patients; in one patient, there was partial obliteration of the malformation. In the group of four patients, embolization had no effect.

Results

Of the 27 patients on whom data are available, 18 were men and nine were women. Their mean age was 35.3 years

at the time of embolization (men, 37.2; women, 31.4). The mean follow-up time was 2.25 years $\pm$ 1.74 months (SD) (range, 3 months to 6 years).

Cerebral hemorrhages did not recur in four of the five patients who had had hemorrhages before embolization. In one patient who had hemorrhages at ages 7, 15, 18, and 20 years, embolization was carried out at age 21 with a good result as determined angiographically. In the ensuing 5 years, she had no further hemorrhages. In another patient, embolization was carried out at age 23 years after a single hemorrhage. It was considered beneficial angiographically, and there were no recurrences in the ensuing 23 months. In the third patient, there had been two hemorrhages at ages 42 and 44 years. Three embolization procedures were carried out with beneficial angiographic results. There were no recurrent hemorrhages in the ensuing 3 years. One patient had a single hemorrhage about 1 month before embolization at age 24 years. Embolization was considered of no angiographic benefit, and there was no recurrence in the 10 months of follow-up. In only one patient did a hemorrhage recur after embolization. The patient had four hemorrhages in 11 years, from ages 29 until 40 years. Embolization was carried out without any beneficial result as determined angiographically. Six months later, the patient had a further hemorrhage. In the next year, there were no further hemorrhages, but there was one episode of weakness of the arm and leg.

Nineteen of the 27 patients were originally seen because of headaches. After embolization, 12 patients reported that their headaches had disappeared or decreased in intensity. In 11 of the 12, embolization was beneficial angiographically. It was also beneficial angiographically in the seven patients whose headaches did not change. In one patient, an initial lessening of the headaches was followed by a worsening; in one patient, the headaches became shorter but sharper; and in five patients, embolization had no effect on the frequency or intensity of the headaches. In no patients were the headaches worse in the initial period after embolization.

Eighteen of the 27 patients were seen initially because of seizures. After embolization, the seizures decreased in frequency or disappeared in 11 patients. However, in seven of the 11 patients, the anticonvulsant therapy after embolization was either different or increased in quantity; in the other four, it was the same, less, or discontinued. In eight of the 11 patients, angiography showed the embolization to be beneficial. In five of the 18 patients, embolization had no effect on the seizures, and, in two of the 18, the seizures were more frequent even though the quantity of medication was increased. In these latter patients in whom the seizures were unchanged or worse, embolization appeared beneficial angiographically in all seven.

In three of five patients who had transient ischemic attacks, the attacks ceased after embolization. In two of these three, embolization was beneficial angiographically. In two patients with transient ischemic attacks, the attacks were as frequent after embolization as before. In one of these two patients, embolization was considered of no benefit angiographically.

Neurologic deficits were present before embolization in

five patients. The quadrantanopsia in one patient disappeared, and, in one patient, weakness and clumsiness of the hand improved after embolization; in both patients, embolization was of benefit as determined angiographically. In two patients, one with paresis of the hand and one with hemiparesis of the hand and leg, the weaknesses progressed over the ensuing months, even though in both the embolization appeared to be beneficial angiographically; one of these patients also had a hemorrhage after embolization. In one patient, there was a progressive deterioration in memory and a change in personality, even though the arteriovenous malformation decreased in size after embolization.

Emboli strayed in 20 patients. There were temporary complications, none longer than 2 days, in six of these 20 patients. Permanent complications occurred in two patients. In one patient with a left hemiparesis, even though embolization was beneficial, the hemiparesis became worse, and a hemianopsia also developed that persisted for 6 months. The second patient had four previous hemorrhages, and, at the time of embolization, a right hemiparesis and right inferior quadrantanopsia were present. Three separate embolization procedures were carried out, all with angiographic benefit. However, immediately after the last procedure, a right homonymous hemianopsia developed that was still present 5 years later.

Discussion

Patients with cerebral arteriovenous malformations are at considerable risk of morbidity or death. The natural history of the untreated disease indicates that 10%–17% will die from hemorrhages and 40%–50% will have their working capacity reduced or will become invalids over the next 20–40 years [4, 9]. In one study of 55 patients, surgical excision of the malformation was carried out with less than 10% morbidity and a 2% mortality. In 44 of the 55 patients (69 procedures), embolization, using particulate barium-impregnated Silastic spheres, was performed with a less than 2% permanent morbidity and no deaths [10].

Cerebral arteriovenous malformations can also be treated by using flow-directed balloon catheters for controlled injection of tissue adhesives. While the latter technique has had notable successes, to our knowledge, a statistical analysis of the results of the technique has not been published, and there have been reports describing the risks of balloon catheterization in patients with cerebral arteriovenous malformations. With the development of safer, less traumatic balloons, the injection of tissue adhesives into the nidus of the malformation may become the procedure of choice.

In our series, five patients had hemorrhages before embolization. In four, the hemorrhages did not recur. The natural history of ruptured arteriovenous malformations indicates that if a patient has had a hemorrhage, there is a one-in-four chance of recurrent bleeding within 4 years [9]. Luessenhop and Presper [11] considered it unlikely that embolization protected a patient from cerebral hemorrhages. The average follow-up in their series was 4 years; in our series, it has been 5, 2, 3, 1, and 1 years (average, 2.4 years). Longer follow-ups of more patients are needed

to establish whether or not embolization protects a patient from hemorrhages.

Embolization appeared to be of considerable benefit in reducing or eliminating the headache in 12 of 19 patients. However, in the seven patients in whom embolization had no effect on the severity of the headaches, embolization was considered beneficial angiographically. Similarly, Luessenhop and Presper [11] found embolization very effective in headache control.

While our data indicate that embolization reduced the incidence of seizures in 11 of 18 patients, in only four patients was the quantity of anticonvulsant medication the same or less after embolization. In three of the four patients, embolization achieved some benefit as determined angiographically (decrease in size of arteriovenous malformation in one, perfusion of normal brain in one, and both decrease in malformation size and perfusion of normal brain in one). These data should be compared with those of five of the 18 patients in whom the seizure incidence was unchanged and with two of the 18 patients in whom, even though the malformation was decreased in size after embolization, the seizure frequency increased. Therefore, our data do not confirm the opinion of Kusske and Kelly [12], who described a major decrease in the frequency and severity of seizures after particulate embolization in six of eight patients. We concur with Luessenhop and Presper [11] that embolization alone does not reduce the incidence of seizures.

The effect of embolization on neurologic deficits seemed varied. Transient ischemic neurologic attacks disappeared in three patients but persisted in two. Similarly, there was a considerable improvement in neurologic deficits in two patients, but progression of the deficits in two others, and progressive mental deterioration in one. In two patients, embolization caused complications. In the Luessenhop and Presper [11] series, embolization arrested progression of the neurologic deficits in eight of 12 patients.

Many patients had a feeling of increased vigor and well-being after embolization. The significance of that is difficult to determine. It either represents a physiologic response or a manifestation of the relief and gratitude felt by the patients after therapy for a potentially catastrophic disease.

To conclude, our data indicate that, in patients with large, surgically inaccessible cerebral arteriovenous malformations, embolization reduces the frequency of headaches but has no beneficial effect on the frequency of seizures or on the development of neurologic deficits. As yet, the effect of embolization on the incidence of recurrent hemorrhages is undetermined.

Addendum

Since acceptance of this paper, results of the treatment of 46 cerebral arteriovenous malformations with intraarterial Bucrylate have been published: Bank WO, Kerber CW, Cromwell LD. Treatment of intracerebral arteriovenous malformations with isobutyl 2-cyanoacrylate: initial clinical experience. *Radiology* 1981;139:609–616.

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