Retrograde thrombosis of feeding arteries after removal of arteriovenous malformations

YOSHI MIYASAKA, M.D., KENZO YADA, M.D., TAKASHI OHWADA, M.D., TAKAO KITAHARA, M.D., MASATAKENDOHO, M.D., MOTOYOSHI SAITO, M.D., AKIRA KURATA, M.D., AND HIROTOSHI OHTAKA, M.D.

Departments of Neurosurgery and Critical Care Medicine, Kitasato University School of Medicine, Sagamihara, Kanagawa, Japan

Five cases of retrograde thrombosis of former feeding arteries after removal of an arteriovenous malformation (AVM) are reported. The clinical features of these patients were studied and compared to those of 71 patients without this complication. The following characteristics were found to correlate with retrograde thrombosis: 1) advancing age of the patient; 2) large AVM size; and 3) markedly dilated and elongated feeders. It is suggested that the slow flow in the former feeding arteries that was observed immediately after AVM removal and pathological changes in these vessels due to long-standing hemodynamic stresses contributed to the development of retrograde thrombosis. Neurological manifestations related to retrograde thrombosis were noted in three of the five cases. Although infrequent, this complication should be considered as a serious possibility following removal of an AVM.

KEY WORDS • arteriovenous malformation • thrombosis • feeding artery • retrograde thrombosis

COMPLICATIONS following removal of an arteriovenous malformation (AVM) have been well documented in the literature. These include brain swelling due to "normal perfusion pressure breakthrough," postoperative hemorrhage from a residual nidus, hemorrhage from insufficient hemostasis with a high-pressure afterload, and brain swelling secondary to impairment of venous circulation. No studies have specifically focused attention on postoperative retrograde thrombosis of the feeding arteries to an excised AVM. The purpose of the present investigation is to clarify the clinical features of patients who developed this complication and to consider factors giving rise to it.

Clinical Material and Methods

The present study group consisted of 76 patients who underwent surgical excision of a cerebral AVM and postoperative angiographic studies. There were 33 females and 43 males, ranging in age from 1 to 70 years (average age 28 years).

The criteria for retrograde thrombosis were: 1) removal of an entire nidus without injury to the feeding arteries and without temporary clipping of these arteries; 2) postoperative serial angiograms disclosing retrograde filling defects of contrast material in former AVM feeders, starting from the point where the arteries entered the malformation; and 3) filling defect exceeding 3 cm in length. An AVM was defined as "large" when its maximum diameter was greater than 5 cm, "medium-sized" when it was between 3 and 5 cm, and "small" when it was less than 3 cm.

The ratio of the maximum diameter of the most dilated feeding artery (FA) to the maximum diameter of the internal carotid artery (ICA) in the cavernous portion (FA/ICA ratio) was calculated, and the distance from the tuberculum sellae (TS) to the AVM (TS-AVM distance) was obtained from lateral angiograms. These measurements were used to estimate quantitatively the degree of dilatation and elongation of the feeding arteries.

Statistical analysis was performed by Statistical Analysis Systems using Student's t-test or Fisher's exact probability test.

Illustrative Case Reports

Case 1

This 70-year-old woman had suffered two intracerebral hemorrhages. Preoperative neurological examination showed mild aphasia and a right hemiparesis. Pre-
operative angiograms revealed a medium-sized AVM in the medial parietal lobe (Fig. 1) that was supplied by a markedly dilated and elongated anterior cerebral artery (ACA). Three days after an uneventful removal of the AVM, the patient noticed increased weakness in the right leg. Thirteen days later, she underwent left carotid angiography. Retrograde thrombosis of the left ACA was recognized from the point where the ACA entered the AVM to where the ACA branched off to the callosomarginal artery (Fig. 2). The proximal part
FIG. 5. Preoperative right carotid angiograms in Case 3 demonstrating a large frontal arteriovenous malformation fed by a markedly dilated and elongated anterior cerebral artery. **Upper Left:** Lateral view, early arterial phase. **Lower Left:** Lateral view, late arterial phase. **Right:** Anteroposterior view, arterial phase.

**FIG. 3.** Angiograms, lateral (left) and anteroposterior (right) views, in Case 2. **Upper:** Preoperative left carotid angiograms showing a large parieto-occipital arteriovenous malformation (AVM) supplied by the feeders arising from a markedly dilated and elongated middle cerebral artery in association with the anterior cerebral artery. **Lower:** Preoperative vertebral angiograms revealing feeding arteries arising from the markedly dilated and elongated posterior cerebral artery.

**FIG. 4.** Postoperative vertebral angiogram in Case 2 showing complete removal of the arteriovenous malformation and retrograde thrombosis in the left posterior cerebral artery (large arrow). The medial posterior choroidal artery (small arrows) was preserved.

of the left ACA demonstrated very slow flow, referred to as “stagnant arterial flow” (Fig. 2).

**Case 2**

This 37-year-old man was admitted to our institution following intracerebral hemorrhage. Preoperative neurological findings included left parietal lobe signs. Admission left carotid and vertebral angiograms showed a large parieto-occipital AVM that was supplied by feeding arteries arising from a markedly dilated and elongated middle cerebral artery (MCA) and posterior cerebral artery (PCA) in association with the ACA (Fig. 3). The patient developed a right homonymous hemianopsia after an uneventful removal of the AVM. Postoperative angiography performed 18 days after the operation demonstrated total removal of AVM. However, a vertebral angiogram revealed thrombosis of the left PCA from its site of entry into the malformation to the origin of the medial posterior choroidal artery (Fig. 4). A computerized tomography (CT) scan demonstrated infarction in the PCA territory.

**Case 3**

This 46-year-old woman presented with frequent psychomotor and generalized tonic-clonic seizures over a 12-year period. Neurological examination at the time of admission was normal. A right carotid angiogram demonstrated a large frontal AVM fed by a markedly dilated and elongated ACA (Fig. 5). Total removal of the nidus was achieved and the patient showed no
Fig. 6. Postoperative right carotid angiograms, lateral (left) and anteroposterior (right) views, in Case 3 showing complete removal of the arteriovenous malformation and retrograde thrombosis in the anterior cerebral artery (arrows). There is good collateral flow from the frontopolar artery (small arrowheads) and the middle cerebral artery (large arrowheads) into the region of the frontal lobe.

neurological deficit after the operation. Right carotid angiography performed 4 days later showed that no contrast material entered the right ACA from its point of entry into the AVM to the origin of the frontopolar artery (Fig. 6). However, there was good collateral flow from the frontopolar artery and the MCA into the frontal lobe region (Fig. 6).

Results

Summary of Cases

Five of 76 patients (6.6%) who underwent removal of an AVM developed retrograde thrombosis. These five patients ranged in age from 37 to 70 years (mean 46 years). The presenting symptoms were seizure in three patients and hemorrhage in two. The AVM affected the frontal lobe region in two patients, the parietal lobe in two, and the parieto-occipital lobe in one. Two AVM's were supplied by the ACA, two by the MCA, and one by the PCA and the MCA in association with the ACA. The maximum diameter of the AVM's ranged from 3 to 6 cm.

The intervals between the operation and the first postoperative angiogram were 4, 8, 13, 18, and 30 days. Retrograde thrombosis was observed only in the parent feeding arteries in all five patients. The ACA was affected in two patients, the MCA in two, and the PCA in one. The thrombosed section of artery ranged from 4 to 9 cm in length. In addition to the retrograde thrombosis in three of the five cases, very slow-flowing vessels, termed "stagnating arteries,"
were observed among the former feeders proximal to the point of the obstruction. This finding was obtained only in cases in which angiography was performed within 2 weeks following the removal of the AVM.

Neurological manifestations secondary to thrombotic infarction occurred in three of the five patients. Postoperative CT scans of these patients showed infarcted areas which were consistent with the neurological deficits.

Comparison of Patients With and Without Retrograde Thrombosis

The mean age of the five patients with retrograde thrombosis was 46.2 ± 13.7 years (± standard deviation), whereas that of the 71 patients without the complication was 28.2 ± 14.9 years. The difference was statistically significant (t-test, p = 0.011), indicating that retrograde thrombosis was more likely to occur in older patients. Retrograde thrombosis developed in three (14%) of the 22 patients without hemorrhage compared with two (4%) of the 54 patients with hemorrhage. The difference was not statistically significant (Fisher's exact probability test, p = 0.142).

Retrograde thrombosis was found in two (18%) of 11 patients with large AVM's and in three (12%) of 26 patients with medium-sized AVM's (Fig. 7); however, the complication was not observed in any of the 39 patients with small AVM's. Thus, operations for medium-sized and large AVM's were associated with a significantly higher incidence of retrograde thrombosis than those for small AVM's (five (14%) of 37 cases vs. none of 39 cases; Fisher's exact probability test, p = 0.024).
Complications after removal of AVM's have been reported to include: brain swelling secondary to "normal perfusion pressure breakthrough;" postoperative intracerebral hemorrhage from a residual nidus; hemorrhage from insufficient hemostasis with a high-pressure afterload; and brain swelling due to a compromised venous circulation. A few reports describing postoperative retrograde thrombosis of a feeding artery are available. Pertuiset, et al., reported the first case of retrograde thrombosis. Their patient had a temporoparietal AVM supplied by a large PCA. At the first operation, two clips were placed on the peduncle segment of the PCA. Postoperative angiograms showed backward thrombosis of the PCA from the clipped portion to the bifurcation of the basilar artery. Unfortunately, a description of neurological deficits was not given. Luessenhop and Rosa reported the second case, associated with a large AVM. Retrograde thrombosis developed in a clipped feeding artery and resulted in severe morbidity. The third case was reported by Solomon and Stein. The AVM was located in the parieto-occipital lobe and was supplied by the PCA. Postoperative retrograde arterial occlusion was demonstrated in the PCA and resulted in homonymous hemianopsia. Malis and Hassler and Steinmetz emphasized the possibility of the development of this complication.

The present study suggested that retrograde thrombosis of former AVM feeders is not a frequent complication. It was observed in only five (6.6%) of 76 patients who underwent postoperative angiography. However, the incidence increased to 14% (five of 37 patients) when the patient population was limited to those with medium-sized and large AVM's. Three of five patients developed postoperative neurological manifestations secondary to thrombosis of the feeding arteries. The development of neurological deficits was dependent upon which vessels were occluded, the available collateral vessels, and the structures affected.

In the present study, statistical analysis disclosed that retrograde thrombosis was more likely to occur in older patients and in those with medium-sized and large AVM's. Other factors contributing to retrograde thrombosis included marked elongation and dilatation of the feeding feeders was an important causative factor of retrograde thrombosis.

Systemic Risk Factors for Retrograde Thrombosis

None of the patients suffered from systemic hypertension, cardiac disease, or diabetes mellitus, which are risk factors provoking cerebral thrombosis. Postoperative reduced systemic blood pressure, hyperventilation, or hemoconcentration, which should be considered risk factors resulting in vascular narrowing or arterial thrombosis, were not noted in any of these five patients. Postoperative CT scans did not demonstrate any massive subarachnoid hemorrhage that subsequently induced cerebral vasospasm.

Discussion

Complications after removal of AVM's have been reported to include: brain swelling secondary to "normal perfusion pressure breakthrough;" postoperative intracerebral hemorrhage from a residual nidus; hemorrhage from insufficient hemostasis with a high-pressure afterload; and brain swelling due to a compromised venous circulation. A few reports describing postoperative retrograde thrombosis of a
arteries proximal to the fistulas consisted of intimal thickening, elastic tissue degeneration, and medial thinning or disappearance of the tunica media. He also noted that histological changes accounting for the findings were similar to those of arteriosclerosis and appeared to have been the direct result of induced hemodynamic stresses. From a clinical standpoint, the AVM feeders proximal to the nidus appear to have been affected by hemodynamic stresses. Therefore, histological changes similar to those described by Stehbens would be expected in the markedly elongated and dilated arteries feeding medium-sized and large AVM's.

The present study revealed that retrograde thrombosis was more likely to occur in older patients. This result also suggested that pathological changes in the AVM feeders due to long-standing hemodynamic stresses form a basis for the development of retrograde thrombosis.

Although retrograde thrombosis of former AVM feeders is not a frequent occurrence, it should be considered a distinct postoperative complication following AVM surgery. The prevention of this phenomenon, however, remains an unresolved problem to which we are endeavoring to find an answer.

References


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Address reprint requests to: Yoshio Miyasaka, M.D., Department of Neurosurgery, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228, Japan.