

# Mass Effect Caused by Clinically Unruptured Cerebral Arteriovenous Malformations

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**OBJECTIVE:** It is generally considered that mass effect caused by arteriovenous malformations (AVMs) is evidence of ruptures. In the present study, the incidence of mass effect in clinically unruptured AVMs was evaluated, and the underlying causative factors and pathophysiological mechanisms were studied.

**METHODS:** Twenty-seven patients with clinically unruptured supratentorial pial AVMs were examined. The majority were suffering from epilepsy, and frontal lobe involvement was revealed in approximately half of the patients. Angiographic studies, computed tomographic scans, and magnetic resonance images were obtained from all patients. Twenty-one patients underwent removal of AVMs. In 10 of the surgically treated patients, intraoperative vascular pressure measurements were obtained before removal of the AVMs.

**RESULTS:** Mass effect was detected in 12 (44%) of the 27 patients. Cortical sulci obliteration (eight patients) and lateral ventricle displacement (seven patients) were frequently noted. The volume of AVMs was significantly larger in patients with mass effect than in those without mass effect ( $P < 0.001$ ). Large dilated venous sac or ectatic veins were observed to be associated with mass effect ( $P < 0.001$ ). In only one patient was mass displacement related to a surrounding massive brain edema. Draining vein pressure in patients with mass effect was significantly elevated as compared to the average value in patients without mass effect ( $22 \pm 5$  versus  $12 \pm 3$  mm Hg) ( $P < 0.01$ ).

**CONCLUSION:** The present study suggests that mass effect is not infrequent in clinically unruptured AVMs. Furthermore, multiple causative factors were detected, including the large size of AVMs, marked draining vein dilatation, and brain edema around the AVMs. Findings also indicated that a pathophysiologically high pressure in the venous drainage system may contribute to mass effect. (Neurosurgery 41:1060-1064, 1997)

Key words: Arteriovenous malformations, Draining vein pressure, Mass effect, Unruptured

Mass effect with unruptured arteriovenous malformations (AVMs) has been reported in the literature only since the beginning of the computed tomographic (CT) scan era (1-4, 6, 7, 11). Kumar et al. (4) first discussed various causes of mass effect in patients demonstrated to have unruptured AVMs by CT scanning. Using magnetic resonance (MR) images, Smith et al. (11) assessed the effect of AVMs on adjacent brain tissue. Most of the articles that have been published, however, focused attention on the morphological characteristics of AVMs, and discussion of the underlying pathophysiology has been limited. In the present study, we reevaluated the incidence of mass effect and the causative factors producing this finding in clinically

unruptured AVMs, concentrating attention on pathophysiological mechanisms.

## PATIENTS AND METHODS

Since the advent of MR imaging, 112 consecutive patients with supratentorial pial AVMs were treated at Kitasato University Hospital between January 1986 and December 1994. Of these patients, the 27 patients with clinically unruptured AVMs were selected for the study.

The patients' ages ranged from 10 to 61 years (average age, 35 yr). Angiograms, CT scans, and MR images were available for all patients, and resection of AVMs was performed in 21

patients. Twenty of the patients were epileptic, one exhibited the cerebral steal phenomenon, and five experienced no symptoms. Clinically silent hemorrhage (old small hemorrhage caused by AVMs) was detected at the time of surgery in seven patients. These patients were nevertheless still included in the present study population, because they experienced no clinical evidence of recent intra-axial hematoma formation. Fifteen AVMs were located in the frontal lobe, four in the temporal lobe, two in the frontoparietal lobe, two in the occipital lobe, two in the thalamus, one in the parieto-occipital lobe, and one in the frontotemporal lobe. AVMs were defined as "large" when their maximum diameter was greater than 6 cm, "medium-sized" when it was between 3 and 6 cm, and "small" when it was less than 3 cm. We noted 10 patients with large AVMs, 11 patients with medium-sized lesions, and 6 patients with small lesions. The size of the AVMs was also calculated in terms of their volume, obtained as the product of their three diameters and 0.52 (10).

Intraoperative vascular pressure measurements before resection of the AVMs were obtained in 10 patients (4 patients with mass effect and 6 without mass effect) to clarify the pathophysiological mechanisms underlying the mass effect, using methods previously described in detail (8).

The statistical methods used were  $\chi^2$  analysis, with Yates' correction for expected values less than 10, and the unpaired *t* test. The results were considered significant at *P* < 0.05.

**RESULTS**

Mass effect caused by AVMs were detected in 12 (44%) of the 27 patients. The affected brain structures are summarized in Table 1. Cortical sulci obliteration and lateral ventricle displacement were frequently observed. Table 2 presents the factors contributing to mass effect. Findings confirmed that the volume of AVMs was one of the important determinants for the production of compression of the surrounding brain tissue. Furthermore, an association with large dilated venous sacs/ectatic veins (Fig. 1) was also observed. Gross displacement was related to surrounding massive white-matter edema in only one patient, who had been included in one of our previous reports (9).

No patients experienced objective neurological symptoms suggestive of the presence of increased intracranial pressure

**TABLE 1. Structures Affected by AVMs Producing Mass Effect**

Mass Effect	No. of Patients
Cortical sulci obliterated	8
Lateral ventricles displaced	7
Lateral ventricles only compressed	3
Third ventricle displaced	3
Pineal gland displaced	2
Choroid plexus displaced	1

**TABLE 2. Factors Contributing to Mass Effect in Arteriovenous Malformations<sup>a</sup>**

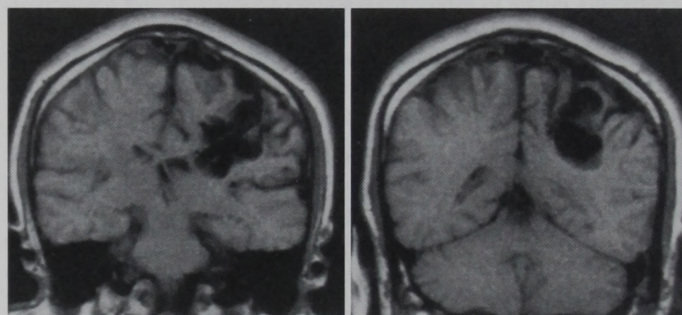
Factors	With Mass Effect (n = 12)	Without Mass Effect (n = 15)
Size of AVMs (maximum diameter)		
Small (<3 cm)	0 <sup>b</sup>	6 <sup>b</sup>
Medium (3–6 cm)	3	8
Large (>6 cm)	9 <sup>c</sup>	1 <sup>c</sup>
Volume of AVMs (mean ± SD; cm <sup>3</sup> )	80 ± 47 <sup>d</sup>	22 ± 22 <sup>d</sup>
Venous sac/ectatic veins	10 <sup>d</sup>	1 <sup>d</sup>
White-matter edema	1	0

<sup>a</sup> Volume of AVMs according to Pasqualin et al. (10). SD, standard deviation; AVMs, arteriovenous malformations.

<sup>b</sup> (*P* < 0.05).

<sup>c</sup> (*P* < 0.01).

<sup>d</sup> (*P* < 0.001).



**FIGURE 1. T1-weighted MR images (coronal views) depicting large AVMs with a dilated venous sac and marked dilatation of the draining vein compressing the ipsilateral lateral ventricle and the cingulate gyrus.**

such as papilledema. The interval from the onset of seizure to admission without anticonvulsants (range, 1 d–20 yr) and the absolute numbers of seizures during these intervals (range, 1 to approximately 50 times) varied from one patient to another. We calculated the numbers of seizures in terms of 1 month, as follows: 17 ± 47 (mean ± standard deviation, n = 9) times per month in patients with mass effect and 6 ± 18 (n = 11) in those without. There was a tendency for the incidence of seizures to be higher in patients with mass effect, but this finding was not statistically significant.

The results of intraoperative vascular pressure measurements are summarized in Table 3. Pressure measurements were obtained in four patients with mass effect and in six without mass effect. No significant differences in central venous pressure were determined between patients with mass effect and those without. Draining vein pressure was, however, significantly higher in patients with mass effect (22 ± 5 versus 12 ± 3 mm Hg) (*P* < 0.01), indicating that this may contribute pathophysiological to the development of compressive effect caused by AVMs.

**TABLE 3. Intravascular Pressure in the Venous Draining System**

Patient No.	Mass Effect <sup>a</sup>	Draining Vein Pressure	Central venous pressure
1	+	29	3
2	+	20	4
3	+	19	3
4	+	18	2
		$22 \pm 5$ mm Hg <sup>b</sup>	$3 \pm 1$ mm Hg
5	-	15	9
6	-	14	3
7	-	13	6
8	-	12	2
9	-	10	4
10	-	6	2
		$12 \pm 3$ mm Hg <sup>b</sup>	$4 \pm 3$ mm Hg

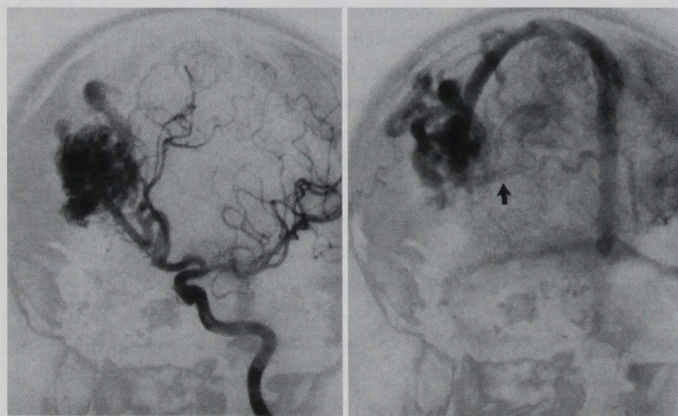
<sup>a</sup> +, present; -, not present.

<sup>b</sup> Statistically significant differences: a) ( $P < 0.01$ ); mean  $\pm$  standard deviation.

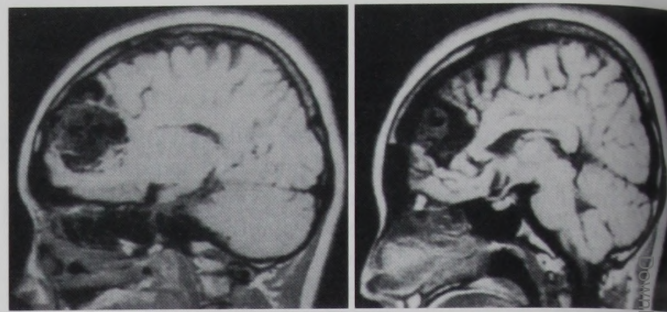
## ILLUSTRATIVE CASES

### Patient 1

A 23-year-old woman presented with tonicoclonic seizure 1 month before admission. Neurological examination revealed no abnormal findings. Left carotid angiography demonstrated medium-sized AVMs (volume, 29 cm<sup>3</sup>) in the right frontal lobe (Fig. 2). The lesion was supplied by the anterior cerebral artery and drained into the dilated cortical veins with a varix (Fig. 2). The reversed flow through the cortical vein was also noted (Fig. 2). MR images revealed compression of the ipsilateral corpus callosum and the cingulate gyrus by the frontal AVMs (Fig. 3). Four months after the onset, total removal of the AVMs was performed and was uneventful. Intraoperative vascular pressure measurement demonstrated a markedly elevated draining vein pressure (29 mm Hg) and moderately elevated sagittal



**FIGURE 2.** Left carotid angiograms (oblique view), arterial phase (left panel) and venous phase (right panel), showing medium-sized AVMs in the frontal lobe fed by the anterior cerebral artery and draining into dilated cortical veins with a varix. Note the reversed flow through the cortical vein (arrow).



**FIGURE 3.** T1-weighted MR images (sagittal view) showing compression of the corpus callosum and the cingulate gyrus by AVMs.

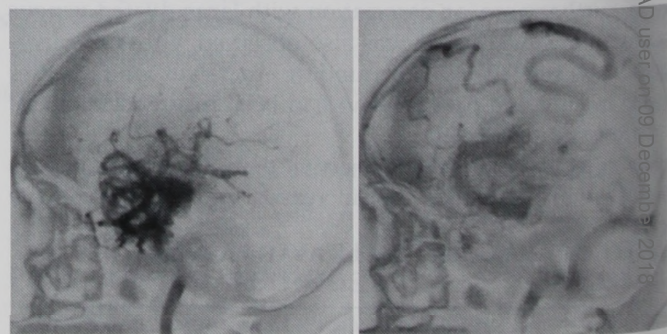
sinus pressure (18 mm Hg) compared to central venous pressure (3 mm Hg).

### Patient 2

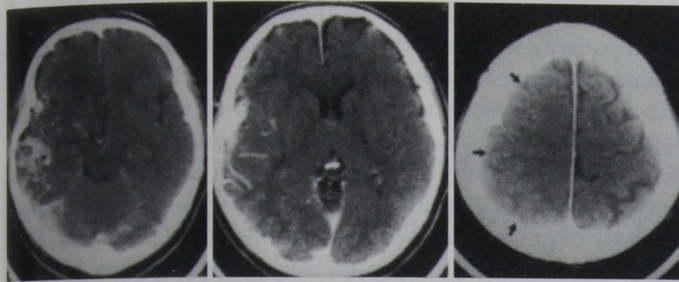
A 44-year-old man presented with frequent focal motor seizures in the right upper limb and face during a 10-year period. Neurological examination at the time of admission revealed mild weakness in the right upper limb. Angiograms of both carotid arteries demonstrated large frontoparietal AVMs (volume, 112 cm<sup>3</sup>) fed by the left anterior and the left middle cerebral arteries. The AVMs drained into markedly elongated and dilated cortical veins. MR images demonstrated compression of the ipsilateral lateral ventricle and the cingulate gyrus caused by large AVMs with a dilated venous sac (Fig. 1). Unfortunately, no intravascular pressure measurement was obtained.

### Patient 3

A 44-year-old man was admitted at our institution because of a temporal lobe seizure experienced 2 months previously. Neurological examination at the time of admission revealed no abnormal findings. Right carotid angiograms revealed large AVMs (volume, 62 cm<sup>3</sup>) situated in the temporal lobe, supplied by branches of the middle cerebral artery (Fig. 4). The AVMs drained into markedly elongated and dilated superior cerebral veins, the superficial middle cerebral vein, and the two anastomotic veins of Labbé (Fig. 4). Preoperative scans demonstrated displacement of midline structures (Fig. 5). Despite the temporal lobe location of the AVMs, obliteration of cortical



**FIGURE 4.** Right carotid angiograms (lateral view), arterial phase (left panel) and venous phase (right panel), showing large AVMs in the temporal lobe supplied by branches from the middle cerebral artery and draining into the markedly elongated and dilated superior cerebral veins, the superficial middle cerebral vein, and the two anastomotic veins of Labbé.



**FIGURE 5.** Contrast-enhanced CT scans (axial view) depicting AVMs in the temporal lobe and the displacement of midline structures (*left and center panels*). Note the obliteration of the cortical sulci in the frontoparietal lobe (*right panel, arrows*).

sulci was depicted not only there, but also in the frontoparietal lobe (Fig. 5). Although a pressure measurement study was not undertaken, it was speculated that a markedly higher venous pressure in a dilated draining vein may have contributed to the development of mass effect.

## DISCUSSION

In the angiographic literature from the pre-CT scan era, AVMs were considered to display mass effect only in hemorrhagic cases. However, since the advent of computed tomography and MR imaging, a number of authors have reported mass effect of unruptured AVMs (1–4, 6, 7, 11). Kumar et al. (4) observed such findings obtained by CT scans for 55% of 60 patients with AVMs. Smith et al. (11) reported compressive effect based on MR images in 40% of 15 lesions. The present study, using both MR images and CT scans showing displacement of surrounding brain structures in 44% of 27 patients, is thus in agreement with earlier investigations in indicating that hemorrhage is not a requirement for causing mass effect of AVMs.

Our analysis of the factors producing and/or contributing to mass effect of unruptured AVMs determined that the volume of AVMs, dilated draining veins, and varices are considered to play an important causative role. Massive edema surrounding the AVMs, although not frequent, can also cause mass effect in unruptured AVMs (9). Kumar et al. (4) similarly concluded that the magnitude of the mass effect largely parallels the size of the AVMs, and enlarged tortuous veins contribute greatly.

The reason why some AVMs cause mass effect and others produce atrophic changes in the surrounding brain tissue rather than compression requires explanation. Fortunately, in the present study, we were able to obtain intraoperative vascular pressure measurements in 10 patients. As a result, we observed the draining vein pressure in patients with mass effect ( $22 \pm 5$  mm Hg) to be significantly higher than in those without ( $12 \pm 3$ ) ( $P < 0.01$ ). We, therefore, speculate that this increase in draining vein pressure, along with the volume of AVMs and the marked draining vein dilatation, may play the determining role.

The clinical significance of mass effect caused by clinically unruptured AVMs is not known (11). However, it seems reasonable that they could play a contributory role in causing

functional derangement of the adjacent brain parenchyma (11). The findings in our study demonstrated that draining vein pressure was significantly elevated in patients with mass effect as compared to the average value in those without mass effect. It is possible that AVMs with venous hypertension may produce venous ischemia with neurological signs or symptoms (5). Thus, AVMs that produce mass effect probably represent a subset with a greater malignant potential, and, therefore, affected patients should be urgently considered for treatment with a combination of preoperative embolization, microsurgery, and radiosurgery before neurological deficits appear. Furthermore, for patients with mass effect, we need to avoid measures at the time of surgery that would further increase cerebral venous pressure.

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## COMMENTS

The authors describe 27 patients with clinically unruptured brain arteriovenous malformations (AVMs); of these, 12 pa-

tients were noted to sustain mass effect. We have observed this phenomena of mass effect caused by AVMs in several patients during the last few years. In general, I agree with the authors that this is caused mostly by increased venous pressure from venous outflow obstruction and relative venous hypertension. The authors were able to measure intravascular pressure in the venous drainage systems of four patients with mass effect from their AVMs and in six patients in whom there was no mass effect. There is a dramatic difference in the venous pressure measured. The authors present the finding, which is becoming increasingly common, that the role of the venous outflow in determining the pathophysiology of AVMs is paramount.

We have observed the treatment of patients who present with AVMs with edema or mass effect to be potentially dangerous. Usually, we have elected to perform intranidal transarterial embolization to reduce arterial inflow through the AVMs, which typically is in a state of severe imbalance, with inflow overwhelming venous outflow. In a few of the patients we have treated, the venous outflow occlusion has been acute. In addition to transarterial embolization, we have used heparin anticoagulation to prevent further venous outflow occlusions.

**Christopher S. Ogilvy**  
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The main focus of this report is to determine what anatomic, physiological, or hemodynamic variables may contribute to the presence of radiological mass effect in unruptured AVMs. The authors evaluated 27 patients harboring AVMs, most of whom presented with seizures. None of the patients presented with clinical hemorrhage. Twenty-one of the patients underwent surgical resection of the AVMs, and in 10 of these, intraoperative vascular pressure measurements were assessed before the resection of the AVMs. The authors detected mass effect in 12 (44%) of their 27 patients, selected by cortical sulci obliteration and lateral ventricular displacement. Radiological mass effect associated with the AVMs was significantly correlated with the larger volume of AVMs and the presence of large dilated venous sacs or ectatic veins. Draining vein pressures in patients with radiographically apparent mass effect were significantly elevated compared to those without mass effect.

The association between radiological mass effect of AVMs and large-sized or enlarged tortuous veins has been previously reported, but its clinical relevance remains uncertain. Although there was a trend toward increasing frequency of seizures in patients with AVMs having mass effect compared with those without mass effect, this difference was not statistically significant, and it is not clear that presentation with seizures is related to radiological mass effect. It is interesting that none of the 12 patients noted as having mass effect from the AVMs were symptomatic with signs of raised intracranial pressure, such as papilledema, headache, or focal neurologi-

cal signs. Of particular note is the authors' novel finding that of 10 patients with intraoperative vascular pressure measurements, draining vein pressures were significantly elevated in those 4 patients with mass effect compared to those 6 patients without mass effect. However, the number of patients involved is small, and other variables, including venous stenoses, the number of draining veins, and the anatomy of arterial feeders, may contribute to determining these pressures. Furthermore, although there is an association between elevated venous pressures, the volume of AVMs, and the presence of dilated or ectatic veins and mass effect, it is not necessarily the case that any of these factors cause or contribute to the radiological mass effect phenomena observed.

**Gary K. Steinberg**  
*Stanford, California*  
**Reid C. Thompson**  
*Los Angeles, California*

This article documents in a small, but well-studied, group of patients what intuitively seems obvious to neurosurgeons, i.e., that cerebral AVMs can be associated with mass effect and that the factors that correlate positively with mass effect are the size and volume of the AVMs, the presence of large venous varices, and the presence of surrounding brain edema. Interestingly, but not surprisingly, in a small group of patients in whom the authors measured the pressure in the draining veins intraoperatively, there was a positive correlation between the presence of mass effect in preoperative studies and the finding of relatively high pressures in the venous drainage system. Whether this was an independent factor, or whether it was related to the finding that these were the larger AVMs, remains speculative.

**Roberto C. Heros**  
*Miami, Florida*

This clinical study of a previously recognized but poorly understood phenomena, i.e., mass effect associated with unruptured AVMs, draws further attention to the previously neglected venous aspect of these malformations whose anatomic and pathophysiological features remain incompletely understood. The authors document a statistical correlation among mass effect, large lesion size, and the presence of venous varices and/or ectasias and have also reported a propensity for elevated venous pressures in such malformations. The clinical relevance of such findings is unknown, although the authors postulate a possible causative correlation between mass effect and seizures; the patient population as defined precludes any current statements regarding hemorrhagic tendencies, etc.

**Duke S. Samson**  
*Dallas, Texas*