Draining Vein Pressure Increases and Hemorrhage in Patients With Arteriovenous Malformation

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Background Recent radiological studies have shown that arteriovenous malformations with impaired venous drainage may be susceptible to hemorrhage. To evaluate this hypothesis using a hemodynamic approach, we measured intravascular pressure during surgery in three patients with arteriovenous malformation.

Summary of Report In three patients we measured intravascular pressures in the draining venous system and the feeding arteries simultaneously before removal of arteriovenous malformations with marked segmental stenotic or occlusive draining veins and evidence of hemorrhage. The draining vein pressures at prestenotic (or preocclusive) sites in the three patients were 38, 25, and 40 mm Hg, respectively, all significantly above the normal cortical venous pressure, whereas pressure measurements in poststenotic sites and the sagittal sinus pressure in the venous drainage system approached normal values. The feeding artery pressures in the patients were lower than normal cortical artery pressure because of the arteriovenous shunt.

Conclusions Intraoperative vascular pressure measurements support the hypothesis that arteriovenous malformations with impaired venous drainage may be associated with a local increase in venous pressure and thus may be susceptible to hemorrhage. (Stroke. 1994;25:504-507.)

Key Words • arteriovenous malformations • cerebral hemorrhage

On the basis of our recent radiological study, we have suggested that the presence of impaired venous drainage (severe stenosis or occlusion of draining veins) may contribute to the development of hemorrhage from arteriovenous malformations (AVMs). However, no hemodynamic studies of AVMs supporting this hypothesis have appeared in the literature. In the present study, we describe three cases in which pressure measurements of the draining veins support the hypothesis that AVMs with impaired venous drainage may be susceptible to hemorrhage.

Case Reports

In the past 4 years we recorded intraoperative vascular pressure measurements in 19 AVMs. In the present study we focused on intravascular pressure measurements in three ruptured AVMs with impaired venous drainage. General anesthesia was induced using fentanyl, pancuronium, and isoflurane and was maintained with these agents and with nitrous oxide. All patients were operated on in the lateral recumbent position, with their heads raised 30 degrees (20 to 25 cm above the level of the heart). Pressure measurements were performed by the same methods that we have previously described in detail. After opening of the dura and before AVM dissection, the intravascular pressures of the feeding artery, draining vein, and sagittal sinus, into which the draining vein flowed, were recorded simultaneously. Needles (26-gauge) were inserted into these vessels with the aid of microscopic visualization. The needles were coupled to a Uniflow disposable pressure transducer (Baxter) with a semi-rigid, fluid-filled tube. The transducer was placed at the same level as that used to measure radial artery pressure (systemic arterial pressure) and central venous pressure (CVP). Both transducers were placed at the level of the right atrium of the heart.

Patient 1

A 17-year-old boy suffered an acute intracranial hemorrhage. A computed tomographic scan demonstrated a massive acute subdural hematoma on the left and a small intracerebral hematoma in the left occipital lobe (Fig 1). Emergency surgery to evacuate the acute subdural hematoma was undertaken. Left vertebral angiograms 7 days and 9 months after hemorrhage revealed a small AVM situated in the left occipital lobe and a marked segmental stenosis in the draining vein (Fig 2). Nine months after the hemorrhage, intravascular pressure measurements and complete removal of the nidus were achieved without complications. As shown in Fig 2, a draining vein pressure (DVP) markedly higher (38 mm Hg) than the CVP (7 mm Hg) was observed at a prestenotic site, and a DVP slightly higher (13 mm Hg) than the CVP was found at a poststenotic site, whereas the sagittal sinus pressure (SSP) and the CVP were the same. The ratio of feeding artery pressure (FAP) (72 mm Hg) to the mean arterial blood pressure (MABP) (89 mm Hg) was 0.81.

Patient 2

A 26-year-old man suffered a visual seizure 4 days before admission. Magnetic resonance imaging 14 days...
after the seizure suggested the presence of an AVM and a small old hematoma cavity in the right occipital lobe (Fig 3). Right carotid angiography 17 days and 7 weeks after seizure demonstrated an occipital lobe AVM and a marked segmental stenosis in the draining vein (Fig 4). The AVM was resected totally without complications 4 months after the seizure. A small old hematoma cavity was observed adjacent to the nidus. As Fig 4 shows, a markedly elevated prestenotic DVP (25 mm Hg), a slightly elevated poststenotic DVP (13 mm Hg), and a slightly elevated SSP (8 mm Hg) were observed in comparison with the CVP (5 mm Hg). The ratio of FAP (57 mm Hg) to MABP (90 mm Hg) was 0.63.

Patient 3

A 37-year-old man suffered an intracerebral hemorrhage due to a small occipital AVM. Computed tomographic scans obtained 2 days after the onset revealed an occipital intracerebral hematoma (Fig 5). Right carotid angiograms 12 days and 1 month after the hemorrhage showed a small occipital AVM and a complete obstruction in the draining vein (Fig 6). Total removal of the AVM and pressure measurements were undertaken without complications 1 month after the hemorrhage. Fig 6 shows that a preocclusive DVP markedly higher (40 mm Hg) than the CVP (4 mm Hg) was observed. The ratio of FAP (51 mm Hg) to MABP (83 mm Hg) was 0.61.

Discussion

Although controversy remains regarding this subject, AVM hemorrhage is thought to be venous bleeding caused by increased pressure transmitted through the arteriovenous shunt.3 Recently, in preoperative angiographic analyses, several authors have suggested that increased resistance in the venous drainage system may contribute to the development of hemorrhage.4-6 We studied the venous drainage system and its impairment in relation to risk of hemorrhage in 108 patients with AVMs, 71 of which were hemorrhagic and 37 nonhemorrhagic.1 Statistical analyses of radiographs demonstrated that AVMs with the following characteristics had a high risk of hemorrhage: (1) one draining vein; (2) severely impaired venous drainage (severe stenosis or occlusion of draining veins); and (3) deep venous drainage alone.1 Thus, it is possible that AVMs with a single draining vein or with impaired venous drainage may be associated with a local increase in venous pressure and thus may be susceptible to hemorrhage.

In a subsequent study we performed intraoperative vascular pressure measurements in 19 AVM patients including previously reported cases2 to clarify any direct causal relation between the venous drainage system and a risk of hemorrhage. DVP was significantly higher in AVMs with hemorrhage (n=13; mean [±SD], 26±7 mm Hg) than in those without (n=6; mean 13±5 mm Hg) (P=.001, unpaired t test). The FAP was significantly higher in the former (60±12 mm Hg) than in the latter (38±4 mm Hg) (P=.0007),2 although still lower than the normal range.7-8 Furthermore, DVP and FAP were inversely related to the number of draining veins.2 These results suggest that a high DVP, as well as a high FAP,
FIG 3. Magnetic resonance imaging 14 days after a seizure in patient 2 showing a low-intensity area in a T1-weighted image (repetition time [TR], 420 milliseconds; echo time [TE], 15 milliseconds; left) and a high- and low-intensity area in a T2-weighted image (TR, 1200 milliseconds; TE, 120 milliseconds; right) located in the right occipital lobe. Old cerebral infarction is noted in the left thalamus.

In the present study we focused on intravascular pressure measurements in three hemorrhagic patients whose venous drainage was severely impaired to clarify any direct causal relation between impaired venous drainage and the development of hemorrhage. Patient 2 in the present study corresponds to case 5 in the earlier report.

FIG 4. Right carotid angiogram 7 weeks after a seizure in patient 2, anteroposterior view, demonstrating a small arteriovenous malformation located in the occipital lobe. The arteriovenous malformation is supplied by the angular artery and drains into the occipital cortical vein. A marked segmental stenosis in the draining vein is shown (arrowhead). Feeding artery pressure (FAP), draining vein pressure (DVP) at a prestenotic site (DVP-1), DVP at a poststenotic site (DVP-2), and sagittal sinus pressure (SSP) during surgery were 57, 25, 13, and 8 mm Hg, respectively. Mean arterial blood pressure and central venous pressure at the time of these pressure measurements were 90 and 5 mm Hg, respectively.

May contribute to the development of hemorrhage from AVMs and support our earlier report that AVMs with only one draining vein are susceptible to hemorrhage.

In the present study we focused on intravascular pressure measurements in three hemorrhagic patients whose venous drainage was severely impaired to clarify any direct causal relation between impaired venous drainage and the development of hemorrhage. Patient 2 in the present study corresponds to case 5 in the earlier report.

FIG 5. Computed tomographic scan 2 days after the onset in patient 3, showing a right occipital intracerebral hematoma.
previous report; patients 1 and 3 have not been described previously.

According to Chimowitz et al. and Little et al., the mean ratio of mean cortical artery pressure to mean systemic arterial pressure in control patients was 0.92±0.10. In the present study the ratios of FAP to MABP in three patients were 0.81, 0.63, and 0.61, which indicated that FAP in AVMs was lower than the normal cortical artery pressure because of the presence of the arteriovenous shunt. Chimowitz et al. and Little et al. reported that normally cortical venous pressure was 1 to 2 mm Hg above CVP. Compared with normal cortical venous pressure, DVP at a prestenotic (or preocclusive) site, in the present study 38, 25, or 40 mm Hg, was markedly elevated, whereas poststenotic DVP and SSP decreased significantly and were close to normal levels. Furthermore, there was a tendency for DVP in the prestenotic sites (or preocclusive sites) in the three patients in the present study (34±8 mm Hg) to be higher than that in the 10 patients (eight previously reported patients and two new patients) with ruptured AVMs without impaired venous drainage (24±4 mm Hg). Our observations indicate that the prestenotic (or preocclusive) measurement sites on the impaired AVM draining veins were exposed to notably high pressure.

We have to regard our results in this study with some caution, because the cortical vein pressure and SSP can vary according to changes in the position of the head and jugular vein compression. However, a high venous pressure was restricted to the prestenotic (or preocclusive) measurement sites in the venous drainage system, which excluded the possibility of an artificially high venous pressure due to technical error.

The marked elevation of DVP in the prestenotic sites (or preocclusive sites) was possibly associated with high resistance in the venous drainage system secondary to stenotic or occlusive changes in the draining veins. Our data support our previous speculation that AVMs with impaired venous drainage may be associated with local increases in venous pressure and thus may be susceptible to hemorrhage.

References


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