Endovascular Management of Cerebral Vasospasm

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KEYWORDS

• Vasospasm • Endovascular • Angioplasty
• Interventional • Subarachnoid hemorrhage
• Angiography

Cerebral vasospasm causes significant morbidity and mortality in patients with subarachnoid hemorrhage (SAH). The management of these patients is challenging and requires the multidisciplinary input of intensive care, neurosurgical, and endovascular specialists. Angiographic vasospasm occurs in approximately 70% of all aneurysmal SAH, but clinical neurological manifestations occur in only one third of these cases.[1] Up to 15% of patients surviving the initial subarachnoid hemorrhage will suffer stroke or death as a result of vasospasm.[2,3] Vasospasm rarely occurs before day 4; it tends to peak at day 7, and it may last up to 2 weeks after the initial hemorrhage.

Most cases of vasospasm can be managed medically. Medical strategies for treating vasospasm include hemodynamic augmentation to improve cerebral perfusion pressure and medical therapy to prevent or reduce cerebral vasospasm. A combination of volume expansion, hemodilution, and induced hypertension (Triple H therapy) has been used extensively, but its value has not been tested rigorously.[4] Currently, oral nimodipine is recommended for patients with aneurysmal SAH. Other, newer agents being evaluated include albumin, statins, magnesium sulphate infusion, and clazosentan (endothelin-1 antagonist).[4] Detailed discussion of medical therapy is beyond the scope of this article. Instead, it will focus on the endovascular therapy of vasospasm and the role of radiological imaging in the appropriate selection of patients who are likely to benefit from this form of treatment.

The clinical diagnosis of vasospasm often is based on detailed neurologic examination. The monitoring of patients at risk for clinical vasospasm requires constant neurological examination by intensive care specialists and the decision making of an experienced, multidisciplinary physician team. The diagnosis of symptomatic vasospasm requires identification of new focal motor deficits or sudden changes in mental status in at-risk patients. These new deficits should not be easily attributed to other causes such as development of hydrocephalus, systemic infection, seizures, or ongoing delirium. Although clinical examination is very useful, it is not always reliable. A significant proportion of patients with SAH may be neurologically impaired or comatose at baseline. In such patients, a meaningful neurological examination may not be obtainable.

IMAGING ASSESSMENT

Diagnostic imaging assessment of a patient with SAH in the vasospasm window serves many functions. These include ruling out other

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pathologies, detecting the presence of vasospasm, and assessing its severity. In a patient with acute neurological deterioration, imaging assessment is essential to triage those patients appropriate for aggressive medical or endovascular therapy. Many different imaging modalities have been used, including transcranial Doppler (TCD) ultrasound, single photon emission computed tomography (SPECT) cerebral blood flow studies, positron emission tomography (PET), magnetic resonance angiography (MRA), magnetic resonance perfusion, stable xenon-enhanced computed tomography (CT), CT angiography, and CT perfusion. In a patient with suspected symptomatic vasospasm, noncontrast CT is a first-line study at most institutions. It can easily rule out other causes for deterioration such as hydrocephalus and rehemorrhage. In addition, a developing hypodensity in the vascular territory of clinical concern could indicate an established infarction. In such patients, aggressive endovascular therapy would be unlikely to be effective, and, in fact, it can be potentially harmful, as it can cause further morbidity or mortality from reperfusion hemorrhage.[5,6] Clearly, the relative size of this infarct needs to be weighed against the benefit of intervening to prevent infarction in a larger area of at-risk parenchyma (the so-called penumbra).[7]

TCD is used in many institutions and has the advantage of being a portable noninvasive study that can be performed at the bedside in the intensive care unit (ICU) setting. The TCD results correlate well with angiographic findings if the vessel under investigation is insonated adequately (**Fig. 1**). Its value, however, is rather limited in patients with poor acoustic windows. The sensitivity of TCD varies depending on the vessel affected by vasospasm, with relatively low sensitivity for supraclinoid internal carotid and anterior cerebral arteries (ACA).[4] TCD has been shown to be specific but not sensitive for vasospasm of the middle cerebral artery (MCA) when compared with angiography, and it is poorly predictive of developing secondary cerebral infarction.[8,9] In addition to limitations imposed by poor acoustic window, the utility of TCD is hampered further by operator dependence and inability to study the distal vessels.

Use of imaging modalities such as magnetic resonance imaging (MRI)/MRA/magnetic resonance perfusion, PET, SPECT, and xenon CT that assess cerebral vasculature or brain perfusion often require the patient to remain still for prolonged periods. These techniques are not universally available and are often not practical for routine clinical use in these very sick patients. In recent years, a combination of CT angiography (CTA) and CT perfusion (CTP) has emerged as an important tool. It is very helpful in triaging patients with suspicion of vasospasm into those who should have aggressive medical management and others who should undergo early endovascular therapy. It is an attractive technique as it is a fast, readily available, relatively inexpensive, and practical imaging modality well suited to ICU patients. This can be combined easily with noncontrast head CT and performed on most commercially available scanners. Modern multidetector scanners are capable of rapidly assessing the caliber of the intracranial arteries using CTA and the brain parenchymal perfusion (CTP) with the use of 50 to 100 cc bolus of iodinated contrast (**Figs. 2** and **3**). Multidetector CTA has a very high accuracy of 98% to 100% for detecting severe vasospasm when compared with digital subtraction angiography.[10–12] Lower degrees of accuracy for mild–moderate vasospasm (57% to 85%) have been reported. Supraclinoid internal carotid artery (ICA) and very distal intracranial arteries are slightly difficult areas to assess on the CTA studies.[10,12] The addition of CTP, however, however improves the accuracy of diagnosis of distal vasospasm by demonstrating tissue-level perfusional abnormalities despite the absence of proximal vasospasm on CTA.

CTP provides several quantitative parameters of cerebrovascular hemodynamics. These include MTT, CBV and CBF.[13] MTT is defined as the average transit time of blood through a given brain region, measured in seconds. CBV is defined as the total volume of blood in a given volume of brain, usually measured in milliliters per 100 grams of brain tissue. CBF is the volume of blood moving through a given volume of brain per unit time, measured in milliliters per 100 grams of brain tissue per minute.

MTT or time to peak (TTP) maps have been shown to be the most sensitive in detecting early auto-regulation changes in cerebral ischemia, and these maps should be interrogated first when reading a CTP study.[10,14] In the authors' experience, if these maps are normal and symmetrical, then clinically significant vasospasm is highly unlikely. Abnormality on these maps, however, mandates close and careful inspection of the CBV and CBF maps to further characterize the severity of the perfusional defect. Three patterns of CT perfusional abnormality can be identified with progressive severity.[10,15,16]

1. Elevated MTT/TTP with normal CBF and normal-to-increased CBV: indicates perfusional abnormality that is adequately compensated for by auto-regulation

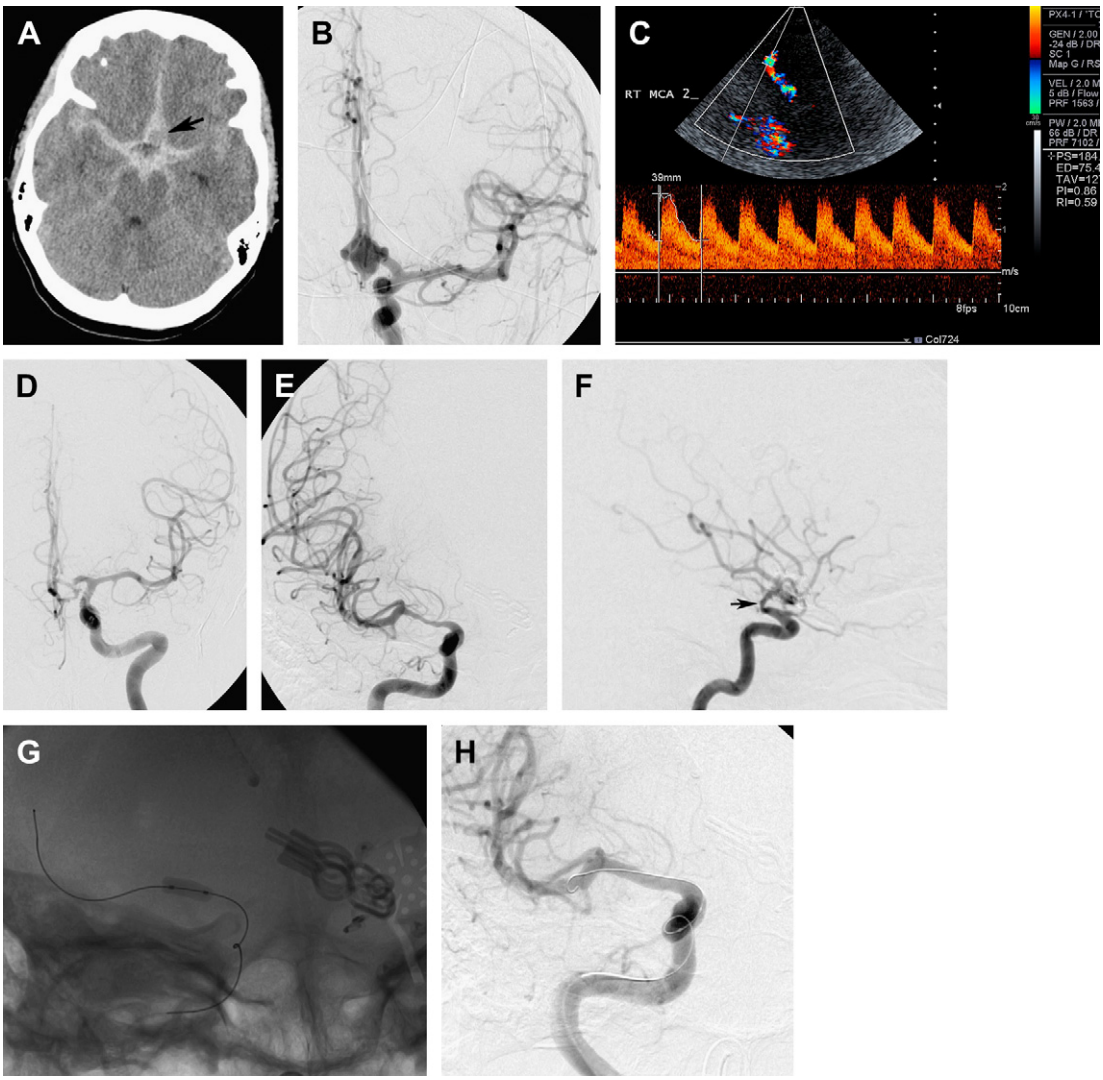

Fig. 1. Utility of Doppler in assessing the vasospasm. A patient with grade 3 SAH from ruptured anterior communicating artery. This aneurysm was clipped. (*A*) Initial CT scan demonstrates a diffuse subarachnoid blood. A small filling defect in the anterior interhemispheric fissure is suggestive of an aneurysm (*arrow*). (*B*) A digital subtraction angiography (DSA) study confirms the presence of a complex anterior communicating artery predominantly opacified from the left internal carotid artery (ICA) injection. Bilateral A2 segments fill from the left ICA injection, and the right A1 segment was hypoplastic or atretic. (*C*) The patient had a waxing and waning course in the ICU. A Doppler study on the sixth day demonstrated findings suggestive of severe vasospasm. This image shows the right middle cerebral artery (MCA) and the peak velocities in this vessel are markedly elevated. (*D*) The left ICA angiogram demonstrates occlusion of the aneurysm and some narrowing of the distal left A1 segment. There is, however, no flow limitation; therefore this vessel was not treated. (*E*) Anteroposterior (AP) and lateral (*F*) angiograms of the right ICA demonstrating severe spasm in the left supraclinoid carotid (*arrow*) and the proximal right MCA. (*G*) Inflation of a hyperform balloon in the right MCA M1 segment during the angioplasty. The patient also underwent ICA angioplasty. (*H*) After angioplasty, the caliber of the ICA and MCA has improved significantly, and there is good augmentation of flow. The patient made a complete recovery.

2. Elevated MTT/TTP with reduced CBF and normal-to-increased CBV: indicates perfusional abnormality with reversible cerebral ischemia (penumbra) (see **Figs. 2** and **3**)
3. Elevated MTT/TTP with reduced CBF and matched reduced CBV: indicates perfusional abnormality with irreversible cerebral ischemia.

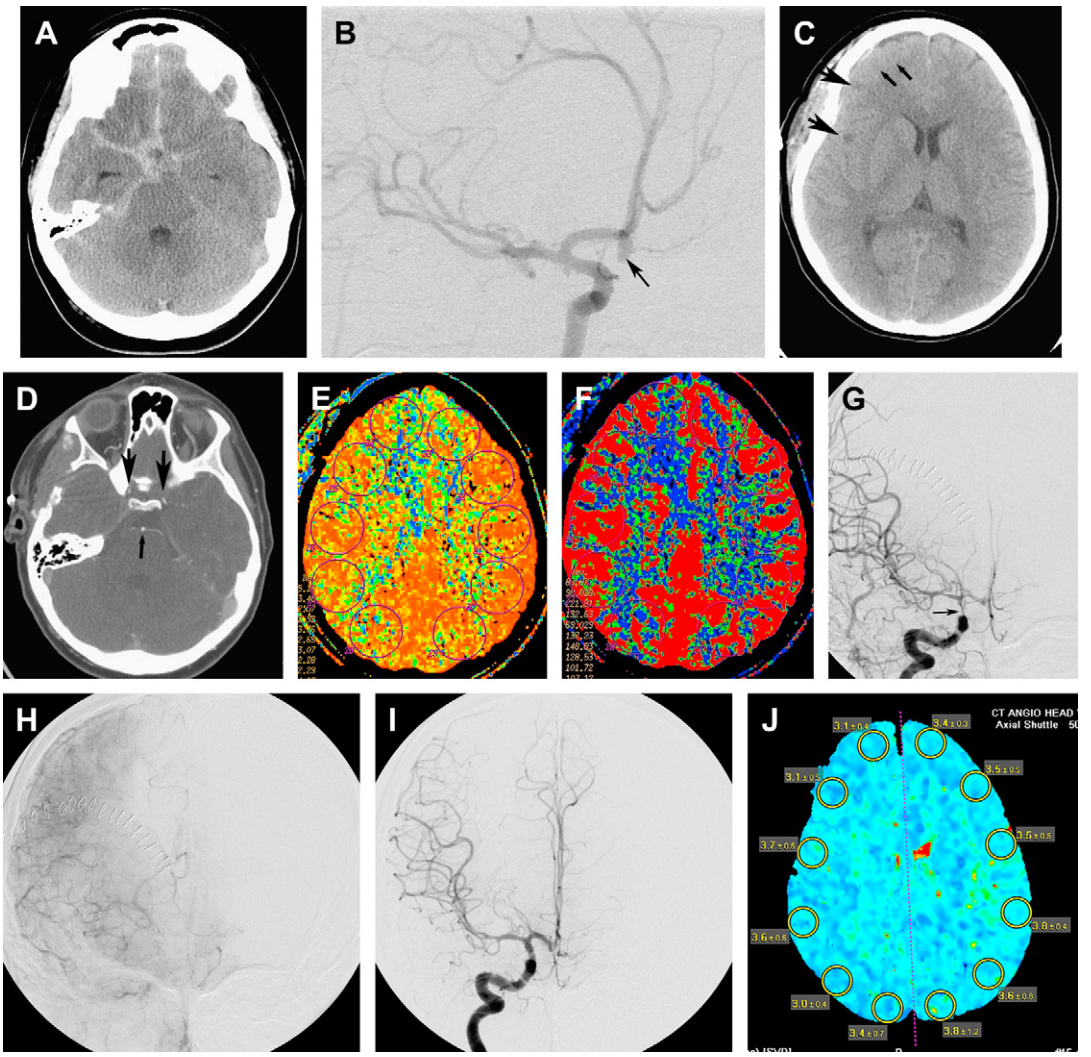

Fig. 2. This middle-aged female patient presented with a diffuse subarachnoid hemorrhage from a ruptured anterior communicating artery aneurysm. (*A*) Initial head CT shows diffuse subarachnoid blood in the basal cisterns. (*B*) An oblique angiogram of the right ICA demonstrates a small, inferiorly pointing anterior communicating artery aneurysm at the junction of the right A1 and A2 segments (*arrow*). This was treated with surgical clipping. (*C*) The patient developed new-onset weakness of left upper and lower extremities on day 8 from the initial SAH. A noncontrast CT demonstrates tiny new hypodensities in the right MCA distribution (*large arrows*) and questionable blurring of gray–white junction in the right frontal region (*small arrows*). (*D*) A CTA study (axial multiplanar reformat) demonstrates severe narrowing of bilateral supraclinoid ICAs (*large arrows*), as well as moderate narrowing of the basilar artery (*small arrow*). (*E*) A CTP study was obtained simultaneously. A mean transit time demonstrates asymmetry between the right and the left hemispheres with prolongation of mean transit times in the anterior cerebral artery (ACA) and MCA distributions (right hemisphere > left hemisphere). (*F*) Corresponding cerebral blood flow maps demonstrate decreased cerebral blood flow, again more severe on the right side. (*G*) A DSA image of the right ICA confirms very severe abnormalities in the caliber of the proximal vessels with profound reduction in caliber of the ICA (*arrow*) and severe narrowing of the MCA and ACA. Similar but slightly less severe abnormalities were present contralaterally (not shown). (*H*) A parenchymal phase of the right ICA angiogram shows heterogenous appearance with paucity of contrast staining, especially in the ACA distribution. The findings of DSA correlate very well with CTP findings. (*I*) After angioplasty, the vessel caliber of the ICA, MCA, and ACA is markedly improved with prompt opacification of the distal branches of ACA and MCA. Similar findings were seen on the left side (not shown). (*J*) A CTP scan the following day (mean transit time [MTT] map shown here) shows reversal of prior abnormalities and symmetrical, normal mean transit times in bilateral hemispheres.

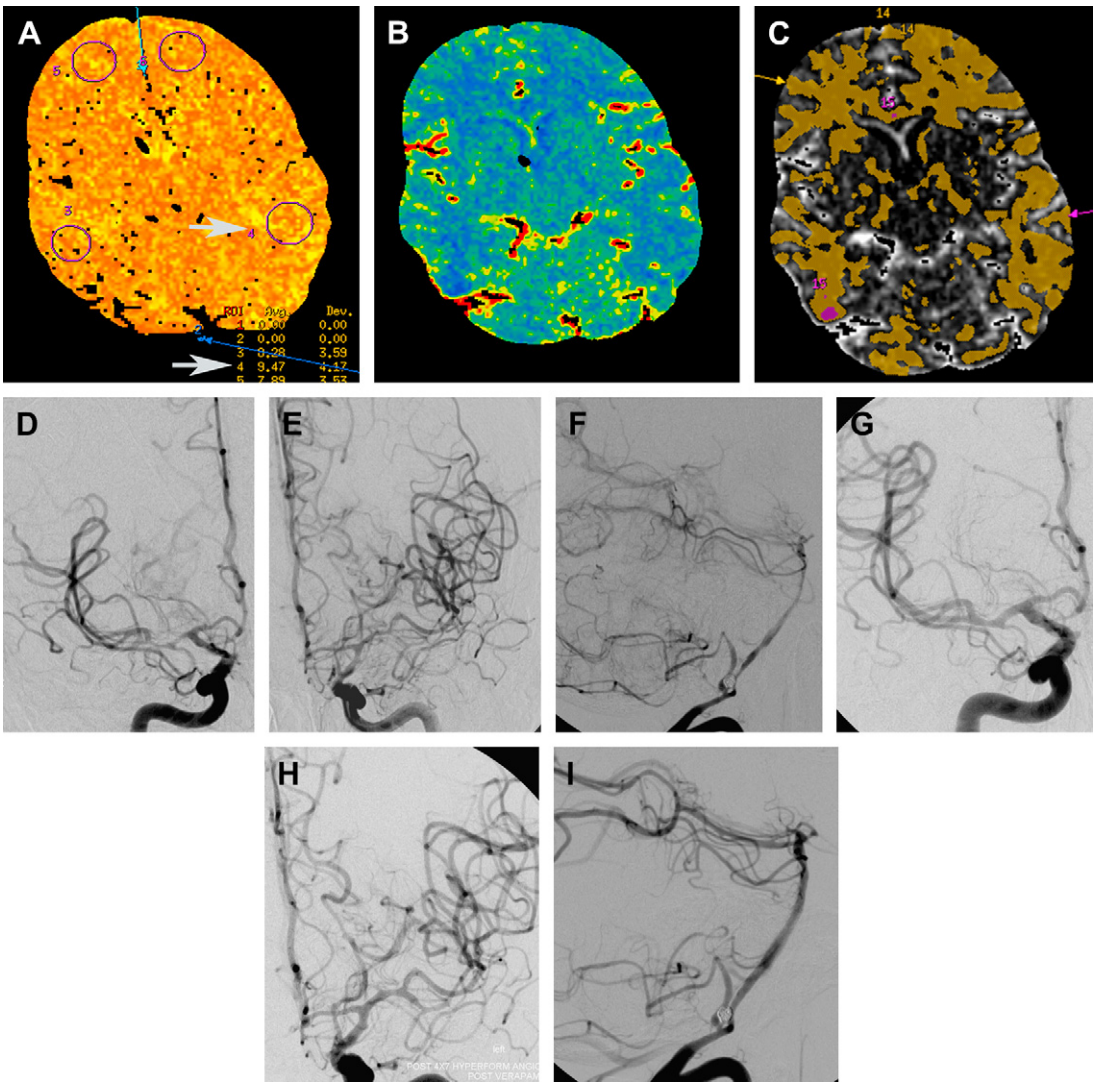

Fig. 3. A patient with diffuse SAH from right posterior inferior cerebellar artery (PICA) aneurysm that was treated with coil embolization. She subsequently developed increased somnolence and right-sided weakness. (*A*) MTT maps reveal bilateral and global prolongation of MTTs for example region of interest 4 (*large arrows*) and show MTT of 9.47 seconds. (*B*) cerebral blood flow (CBF) maps show similar global reduction in CBF. (*C*) Tissue classification map shows areas of reduced CBF but preserved cerebral blood volume (CBV) in yellow (representing ischemic penumbra) and areas of reduced CBF with significantly reduced CBV in purple (indicating likely irreversible ischemia). This map therefore shows that most of the brain is potentially salvageable ischemic penumbra, and aggressive intervention is indicated. Right internal carotid (*D*), left internal carotid (*E*), and vertebral angiograms (*F*), respectively show severe proximal vasospasm involving supraclinoid ICA bilaterally, M1 segments bilaterally, left A1 segment, V4 segment vertebral artery, basilar artery, and proximal posterior cerebral arteries bilaterally. (*G, H, I*) Corresponding angiograms following angioplasty of all involved segments shows marked improvement in caliber. Apart from some small cerebellar infarcts, the patient made an excellent recovery.

This constitutes a relative contraindication to aggressive therapy. Endovascular treatment targeted to this area is not advisable, as this region likely will progress to established cerebral infarction and will be at risk for reperfusion hemorrhage.

MTT and TTP have been shown to be sensitive and early predictors of secondary cerebral infarction in patients with vasospasm.[9,11,17] These CT perfusion changes occur a median of 3 days prior to the development of established infarct on noncontrast CT.[9] Wintermark and colleagues found MTT to have a negative predictive value for cerebral vasospasm of 99% and that the combination of CTA with an MTT threshold of

greater than 6.4 seconds was the most accurate in the diagnosis of cerebral vasospasm.[11] In addition, a cortical regional CBF value of less than 39.3 mL/100 g/min was the most accurate (95%) indicator for the need for endovascular therapy.

Although CTA and CTP are excellent imaging tools, they also have a few limitations. Current limitations include metallic artifact from coils or clips preventing evaluation, problems with contrast bolus timing, and restricted range of parenchymal coverage on perfusion maps. Although the posterior fossa is usually not included on CTP, a range usually can be selected that includes a large part of all three supratentorial vascular territories. Further widespread availability of the latest multidetector scanner technology (256 and 320 slice scanners) will allow complete brain coverage. In addition, the problem of metallic artifacts is being addressed with new, dual-source CT technology.

Patients that are triaged as candidates for endovascular therapy will undergo initial emergent catheter angiography. Vasospasm found on angiography typically is divided into proximal and distal. Most literature divides severity of vasospasm arbitrarily into mild, moderate, and severe based on varying degrees of stenosis.[10,12,18] A useful example is that described by Kassell and colleagues,[18] with four grades: no stenosis or mild (<50%), moderate (50%), and severe (>50%) stenosis. The location of vasospasm determines the method of endovascular treatment employed. Proximal vasospasm should be treated with balloon angioplasty whenever possible. Intra-arterial (IA) vasodilators are used for distal spasm that is not amenable to balloon angioplasty or for vessels considered not safe for angioplasty, for example vasospasm in a vessel segment recently treated with surgical clipping of an aneurysm. The authors also use IA vasodilators as a complement to angioplasty.

INTRA-ARTERIAL INFUSION OF VASODILATORS

Papaverine and calcium channel blockers have been used extensively for IA infusion for pharmacological treatment of cerebral vasospasm. In general, the efficacy of these agents is modest at best and usually of limited duration. For these reasons, their use generally is limited to predominantly distal vasospasm that cannot be treated with angioplasty, mild proximal spasm where angioplasty is not indicated, and other instances where angioplasty is not possible because of anatomical or technical factors. In addition, vasodilators may be used prior to angioplasty to reduce vasomotor tone of the vessel. In the authors' experience (Gandhi D, unpublished data, 2004–2009), the use of calcium channel blockers prior to angioplasty decreases the risk of acute vessel rupture. IA calcium channel blockers are probably the most widely used agents currently in the United States.

Papaverine is an alkaloid of the opium group, and it has been used for a long time as a nonspecific vasodilator in vasospasm via a direct action on smooth muscle. Its use now is largely of historical interest, with most operators preferring the more favorable safety profile of calcium channel blockers. Papaverine had a reported angiographic improvement of around 75% with a modest clinical improvement of 25% to 52%.[6,18,19] The use of papaverine largely has been abandoned because of the common recurrence of vasospasm requiring repeated treatment and complications reported with its use including

- Raised intracranial pressure (ICP)
- Seizures
- Hypotension
- Transient brainstem depression
- Worsening of vasospasm
- Monocular blindness if infused proximal to ophthalmic artery origin.[6,19,20]

IA nimodipine is not available in the United States, but it has been used throughout Europe and Australasia. In a study by Biondi and colleagues[21] of 25 patients with symptomatic vasospasm, there was clinical improvement in 19 (76%) with IA nimodipine. After follow-up of 3 to 6 months, 18 patients (72%) had a favorable clinical outcome. Successful dilatation of infused vessels, however, occurred in only 13 out of 30 (43%) procedures, raising some question as to the cause and effect. The dose used is up to 3 mg per vascular territory at a rate of 1 mg over 10 to 15 minutes to minimize hypotension. Verapamil is another agent used in some centers, although it has been less effective than nicardipine in reversing angiographic vasospasm in the authors' experience (Gandhi D, unpublished data, 2004–2006). Feng and colleagues[22] reported neurological improvement in 29% of 17 patients with vasospasm treated with an average dose of 3 mg. The safety profile was excellent, with just minimal reduction in mean arterial pressure (average of 5 mm Hg) and no evidence of raised intracranial pressure. There is some experimental evidence that demonstrates that nicardipine has greater efficacy than verapamil for endothelin-induced vasospasm.[23]

Nicardipine is a dihydropyridine calcium channel blocker that has more selective effects on vascular smooth muscle than cardiac muscle. Badjatia and

colleagues[24] reported on use of IA nicardipine in 18 patients with vasospasm. There was angiographic and TCD improvement in all patients. Clinical improvement occurred in 42% of patients, and clinically significant improvement in TCD parameters was sustained for 4 days. There was one adverse event where there was an increase in ICP leading to termination of the nicardipine infusion. A series of 20 treatments in 11 patients by Tejada and colleagues[25] showed effective angiographic response in all patients (defined as 60% increase in arterial diameter of the most severely decreased in caliber vessel compared with the very first angiographic run). There was clinical improvement in GCS or resolution of focal symptoms in 10 of 11 (91%) patients. Linfante and colleagues[26] reported on the use of IA nicardipine for 22 patients with symptomatic vasospasm refractory to medical management. They found a 95% significant angiographic improvement rate in these patients. A recent study has confirmed significant improvement in CT perfusion parameters of CBF and MTT following IA treatment with nicardipine.[27] Some previous literature showed discordance between clinical and angiographic outcome following endovascular treatment. The use of CT perfusion for assessing treatment effect may be helpful in these situations, as it is able to show perfusional changes at the microvascular and parenchymal level. These changes may not be evident on inspection of the larger, more proximal arteries using conventional angiography.[21,27,28]

IA nicardipine is currently the drug of choice at the authors' institution. The authors closely monitor the arterial pressures, heart rate, electrocardiogram, and oxygen saturation during the nicardipine infusion. The cerebrospinal fluid (CSF) waveform and CSF pressures also are recorded for the patients who have a ventriculostomy catheter in place. The authors administer the drug very slowly (0.5 to 1 mg/min) and titrate the dose carefully with the arterial pressures during its infusion. If a drop of mean arterial pressure greater than 15 mm Hg or systolic pressure drop of greater than 25 mm Hg is identified, the infusion is halted temporarily. Temporary cessation of infusion often results in gradual return of the arterial pressures to the baseline values, when the infusion can be resumed. A control angiogram is obtained after 3 to 5 mg of the agent has been infused in the affected territory. Modest reduction in blood pressure has been reported with the use of IA nicardipine, and vasopressor support occasionally is needed. Reported systolic blood pressure reductions have been between 10% and 35% or mean systolic reductions of 17 to 23mmHg.[25,26,29] Duration of effect seems favorable when compared with other IA agents, although retreatment is required in some patients.[25]

TRANSLUMINAL BALLOON ANGIOPLASTY

Zubkov and colleagues[30] first described the use of angioplasty in cerebral vasospasm in 1984. The use of this technique has increased steadily over the last two decades with introduction of dedicated neurovascular balloon technology. These balloons can be navigated distally because of their improved trackability and improved safety profiles. Angioplasty is the only method of endovascular treatment that has been shown by numerous subsequent retrospective case series to produce durable clinical improvement. This, however, comes at the cost of needing a specialist with neuroendovascular skills and the small risk of additional serious complications.

Vessels amenable to angioplasty are the proximal intradural arteries, including
Supraclinoid ICA
MCA (M1 and M2 segments)
ACA (A1 and less commonly A2 segments)
Intracranial segment of vertebral arteries (VA)
Basilar and posterior cerebral artery (P1 and P2 segments) (see **Fig. 3**).

It is essential to review the prior (baseline) angiograms in detail before considering an angioplasty for intracranial vasospasm. On the prior studies, careful assessment of vessel morphology and diameter should have been performed. Special note should be made of segments that are congenitally hypoplastic (most commonly A1 segments and intradural vertebral arteries) lest they are confused with arteries affected with vasospasm. Inflating a balloon in congenitally hypoplastic vessels can result in acute vessel rupture. This catastrophic complication can be prevented by review of the prior studies.

As far as possible, softer, dedicated intracranial balloons should be used for intracranial angioplasty to minimize the possibility of vessel rupture. The authors' preference is to use a conformable balloon like Hyperglide (ev3 Endovascular Incorporated, Plymouth, MN, USA) or Hyperform (ev3 Neurovascular, Irvine, CA, USA). These balloons are extremely trackable and can be navigated over a very atraumatic, 0.010 in X-pedion microwire (ev3 Neurovascular). A slight disadvantage of this balloon system is the lack of a continuous flush through the balloon and occasionally problems with steerability in tortuous arteries that form acute angles with more proximal vessels (eg, into the A1 segment). In difficult cases,

a microcatheter can be placed first in the vessel to be angioplastied. This microcatheter is then exchanged for the balloon over a 0.010 in exchange length X-Celerator microwire (ev3 Neurovascular). Occasionally, the authors have used the small-diameter Gateway balloon system (Boston Scientific, Natick, MA, USA) for patients with difficult anatomy. New angioplasty armamentarium is likely to be available in the near future with an increasing range of available sizes of balloons, as well as introduction of newer, over-the-wire balloons. One always should keep in mind that the goal of angioplasty is to improve vessel caliber to augment flow rather than to achieve a picture-perfect result. The authors tend to slightly underinflate the balloon compared with projected normal diameter of the vessel. This gives an additional safety mechanism and decreases the possibility of acute rupture of a vessel. As a rule, the authors do not inflate a balloon at the site of recent surgical clipping of an aneurysm. Fatal rupture of a vessel has been reported by others during angioplasty close to a clipped aneurysm.[31]

A recent review of the literature found reports in the English language on 530 patients undergoing angioplasty for SAH-related vasospasm. Of these 530 patients, 62% improved clinically, with a range of 11% to 93%.[32] The largest series is by Eskridge and colleagues,[33] who reported on retrospective series of 50 consecutive cases (170 vessel segments) using a silicone microballoon (Target Therapeutics, Incorporated, Fremont, CA/Boston Scientific, USA). A significant proportion of patients (61%) showed sustained early neurological improvement within 72 hours, while 6% deteriorated. Two patients (4%) died immediately from vessel rupture. Other larger series have been by Bejjani and colleagues (31 patients with 72% neurological improvement), Higashida and colleagues[5,7,34] (28 patients with 61% neurological improvement), and Fuji and colleagues (19 patients with 63% neurological improvement). Firlik and colleagues[28] showed a 92% clinical improvement rate in 13 patients and also demonstrated quantitative improvement in CBF following angioplasty on Xenon-CT. Other studies have reported lower rates of clinical improvement, but some of these are confounded by the combined use of papaverine and angioplasty.[35,36] A more recent retrospective series of 38 patients by Jestaedt and colleagues[37] showed a clear benefit of angioplasty for reduction in CT evidence of infarct; however, clinical outcome was not assessed. They angioplastied the terminal ICA or MCA in 57 vessels but used 61 untreated anterior cerebral arterial segments with severe spasm as the control. Infarction by CT occurred in 7% of MCA territories compared with 38% of ACA territories.

Similar to acute stroke treatment, endovascular management of vasospasm is time-critical. Prompt referral, assessment, and intervention are essential if angioplasty is to achieve maximum clinical benefit. In one series, 71% of patients angioplastied within a 2-hour window showed sustained clinical improvement compared with 40% in the group treated beyond the 2-hour window, despite both showing good initial angiographic improvement.[38] In addition, Bejjani and colleagues[7] found a higher chance of dramatic clinical improvement in their series if angioplasty was performed within 24 hours of neurological deterioration. Prophylactic balloon angioplasty has been proposed, but it has failed to show a statistically significant improvement in clinical outcome at 3 months in Fisher grade 3 SAH.[39]

Complications of balloon angioplasty include catastrophic vessel rupture, thromboembolism, and reperfusion hemorrhage into an infarct.[5,28,30,31,33] The vessel rupture rate varies from 0 to 7.7%, with an average of 1.1% and major complications overall in up to 5%.[32] The improvements in compliant balloon technology likely mean that the risk of vessel rupture today is in the lower end of this reported range. The decision to angioplasty for vasospasm in the setting of an untreated ruptured aneurysm requires careful consideration of the small risk of rehemorrhage against severity of the vasospasm and the possibility of performing coiling of the aneurysm at the same time as angioplasty.[7,33]

SUMMARY AND RECOMMENDATIONS

Cerebral vasospasm causes significant morbidity in patients with SAH who survive the initial ictus. Prompt imaging evaluation and institution of therapy can be highly effective in improving outcomes in these sick patients. Endovascular therapy should be used early and emergently in those shown on imaging triage to have findings suggestive of severe vasospasm or perfusion impairment despite medical management. Perfusion scans, if available, should be assessed carefully for evidence of significant established irreversible ischemia. For proximal vasospasm, wherever technically possible, balloon angioplasty should be used, as this is the only method that shows durable clinical improvement. IA vasodilators are used for distal vasospasm and as an adjunct to angioplasty. The sustained efficacy of IA vasodilators, however, is less well established, and repeated treatments may be necessary.

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