Vasospasm Secondary to Ruptured Aneurysm: Assessment by Digital Intravenous Angiography

Richard S. Pinto,1 Irvin I. Kricheff,1 Gary De Filipp,1 Eugene S. Flamm,2 and Joseph P. Lin1

Digital intravenous angiography was used for the documentation and evaluation of cerebral vasospasm secondary to acute ruptured aneurysm. Attenuation of vessel caliber and/or generalized poor visualization of intracranial arteries and prolongation of circulation time were observed in seven patients with the acute clinical diagnosis of cerebral vasospasm. Posttreatment intravenous angiography demonstrated improvement in both arterial caliber and circulation time in six patients who responded to medical therapy for vasospasm. Digital intravenous angiography allowed repeat investigations of the intracranial vasculature safely and quickly without undue risk to the critically ill patient.

Rupture of an intracranial aneurysm may severely reduce the caliber of intracranial arteries and often leads to cerebral ischemia, infarction, and clinical deterioration of the patient before surgical ligation of the offending aneurysm [1–3]. A presumptive diagnosis of vasospasm is usually made in a patient who clinically deteriorates preoperatively and in whom computed tomography and/or lumbar puncture has excluded a rebleed. Most investigators require cerebral arteriography for documentation of vasospasm before instituting medical therapy [4–6]. We performed digital intravenous angiography to assess intracranial arterial caliber and to measure circulation time in seven patients who clinically worsened in their preoperative course.

Subjects and Methods

A total of 16 patients were evaluated. Nine patients were examined only once, while seven patients were examined twice. None of the nine patients in the former group clinically worsened during their preoperative course, although in seven patients various degrees of vasospasm were demonstrated on the initial diagnostic arteriography. In these patients intravenous angiography was used to confirm absence of vasospasm immediately before surgery. The other two patients in this group were examined after surgery to pinpoint aneurysm clip location.

This report concerns the seven patients who were examined twice, initially after the onset of clinical deterioration and again after a course of medical therapy for vasospasm. Four of these seven patients were treated for severe bilateral diffuse vasospasm with aminophylline and isoproterenol. The other three patients clinically demonstrated unilateral vasospasm and were treated with volume expansion or hypertension. The mean interstudy interval for patients studied twice was 8 days.

Intracranial arterial caliber was assessed on both intravenous angiograms in all seven symptomatic patients. Circulation time (interval in seconds between the initial filling of the carotid siphon and the initial filling of an ipsilateral parietal vein) was measured for each cerebral hemisphere on both angiograms in all seven patients. Intravenous angiography was performed using a Philips digital vascular imager. A Teflon 5.5 French catheter, 65 cm long with 10 side holes (Universal Medical Instrument Corp., Ballston Spa, NY) was used for all studies. The catheter was placed in the superior vena cava adjacent to the right atrium. Renografin-76 (40 ml/series) was injected at a rate of 15 ml/sec at 31.6 kg/cm.

In 14 patients a 23 cm image-intensifier field was used and images were obtained at two frames/sec. In the other two patients in our series and subsequently, a 14 cm image-intensifier field was used. All images were reprocessed to obtain the best possible image. Although both anteroposterior and lateral projections were obtained, all data in this study pertaining to arterial caliber and circulation time were derived from the anteroposterior view.

Results

Six of the seven deteriorated patients showed marked clinical improvement within 24 hr of the start of therapy. One patient showed no clinical improvement. In all seven symptomatic patients the initial intravenous angiogram demonstrated narrow arterial caliber or generalized poor visualization of intracranial arteries with slow progression of contrast medium through distal sylvian opercular vessels, indicative of vasospasm. In four of seven patients the initial intravenous angiogram documented diffuse vasospasm with involvement of both cerebral hemispheres (fig. 1). In the diffuse vasospasm group there were two ruptured anterior communicating artery aneurysms and two ruptured basilar tip aneurysms. In three patients the initial angiogram documented unilateral vasospasm with attenuation of arterial caliber, primarily involving sylvian opercular vessels unilaterally (fig. 2). Circulation time was different from one hemisphere to the other in this group of patients, which comprised two middle cerebral artery (MCA) aneurysms and one posterior communicating artery aneurysm.

In the diffuse vasospasm group the slowest initial circulation times were noted in patients who clinically demonstrated the most severe aspects of cerebral ischemia, including marked bilateral motor paresis and severe alteration in mental status. (table 1, cases

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TABLE 1: Intracranial Circulation Time in Patients with Vasospasm

<table>
<thead>
<tr>
<th>Group: Case No.</th>
<th>Time (sec)</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse vasospasm:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>7.5</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>8.0</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>NM</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>5.0</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Unilateral vasospasm:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>4.0 (R)</td>
<td>3.0 (R)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>3.0 (L)</td>
<td>3.0 (L)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2.5 (R)</td>
<td>3.0 (R)</td>
<td></td>
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<tr>
<td>3</td>
<td>3.5 (L)</td>
<td>3.0 (L)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>4.0 (R)</td>
<td>3.5 (R)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>3.5 (L)</td>
<td>3.5 (L)</td>
<td></td>
</tr>
</tbody>
</table>

Note.—NM = not measured; R = right hemisphere; L = left hemisphere.

1 and 2). In one patient with severe diffuse vasospasm, initial circulation time could not be measured because of motion misregistration artifacts in the venous phase of the serial angiogram. In the unilateral vasospasm group all three patients demonstrated altered mental status and two of three patients showed mild contralateral hemiparesis. Initial circulation times in each case reflected dissociation between the two cerebral hemispheres (table 1, cases 5–7).

All seven patients were reexamined 7 or 8 days after the initial intravenous angiogram. Clinical improvement was seen in six patients. Of the four patients with diffuse vasospasm on the initial study, three demonstrated improvement in arterial caliber and two had improved circulation times on the second intravenous angiogram. The patient whose initial circulation time could not be measured had an abnormal circulation time on the follow-up study (table 1, case 3). This patient showed no clinical improvement after medical therapy and subsequently suffered two rebleeds and died. In the three patients whose initial angiograms demonstrated a
unilateral circulatory defect, resolution of vasospasm was observed on the second intravenous angiogram, in which circulation times for the two cerebral hemispheres were equal in each case (table 1, cases 5–7).

Discussion

Digital intravenous angiography has a prominent place in the workup of patients with atherosclerotic cerebrovascular disease [7–10]. We used digital subtraction intravenous angiography to evaluate cerebral vasospasm secondary to ruptured intracranial aneurysm.

The diagnosis of vasospasm is made by observing attenuation of arterial caliber of one cerebral hemisphere (unilateral vasospasm) or a generalized poor visualization of all intracranial vessels (diffuse vasospasm). Resolution of vasospasm can be determined by comparing posttreatment angiograms with data from the initial study. Measurement of circulation time [11] and its variation from normal is an ancillary radiologic indication of vasospasm. In patients with unilateral vasospasm a circulatory defect can be demonstrated by observing delay in contrast-media opacification of ipsilateral intracranial vessels and by documenting dissociation in circulation times between cerebral hemispheres. Resolution of vasospasm as determined by arterial caliber on posttreatment angiograms can be confirmed by comparing pre- and posttreatment circulation times. Cerebral circulatory defects from unilateral extracranial carotid stenosis or generalized poor cardiac output can be circumvented by obtaining posttreatment comparison data.

Digital intravenous angiography is expeditious in providing answers to clinical questions; it requires little angiographic expertise, since no selective vascular catheterization is necessary; and it is quickly performed because of the ability of real-time image subtraction. The procedure probably involves less risk to the patient than conventional cerebral arteriography. Digital intravenous angiography is not recommended for the detailed investigation of the vascular anatomy that is required for the primary diagnosis and pretreatment assessment of intracranial aneurysms. However, it plays an important role in the documentation and evaluation of cerebral vasospasm and of its subsequent resolution prior to surgical ligation of an aneurysm.

REFERENCES

3. Crompton MR. Cerebral infarction following the rupture of cerebral berry aneurysms. Brain 1964;87:263–280