The Predicted Value of Arteriography in Nontraumatic Intracerebral Hemorrhage

GILBERT J. TOFFOL, D.O.,* JOSÉ BILLER, M.D.,* HAROLD P. ADAMS, JR., M.D.,* AND WENDY R.K. SMOKER, M.D.†

SUMMARY We retrospectively assessed the diagnostic value of cerebral arteriography for the search of an etiology in 102 patients with nontraumatic intracerebral hemorrhage evaluated between 1980 and 1985. Arteriography was diagnostic in 22 of 50 non-hypertensive patients and in only 6 of 47 hypertensive patients. Five patients with a bleeding diathesis had normal arteriography. From the total group, we found 12 saccular aneurysms, 9 arteriovenous malformations, 3 cases of moyamoya and 3 instances of superior sagittal sinus thrombosis. One patient had metastatic choriocarcinoma.

Sites of hemorrhage among all patients with diagnostic arteriograms were: lobar 19, intraventricular 5, thalamic 2, caudate 1, and corpus callosum 1. Lobar hemorrhages in the non-hypertensive group and intraventricular hemorrhages in hypertensive individuals had the highest yield of arteriographic abnormalities. We believe cerebral arteriography is indicated in non-hypertensive patients with lobar hemorrhages. Most hypertensive patients, in particular those with putaminal hemorrhages, do not require arteriography.

COMPUTED TOMOGRAPHY (CT) is the best diagnostic modality for the evaluation of intracerebral hemorrhage (ICH).1 CT determines both the size and location, as well as, suggests an etiology of the ICH. Hayward and O’Reilly2 claim that, even without clinical information, CT predicts the etiology of nontraumatic ICH with an accuracy of 90%. Prior to CT, cerebral arteriography was commonly used for the evaluation of these patients.3,4 However, arteriography failed to differentiate mass effect of a hematoma, from that of cerebral infarction or neoplasm.3,5,6

To predict the diagnostic value of cerebral arteriography for the search of an etiology in patients with nontraumatic ICH, identified by CT, we retrospectively reviewed the clinical records, angiography results, and CT examinations of patients with ICH evaluated between 1980 and 1985.

Patients and Methods

Our patient population was selected by reviewing the arteriographic records from the University of Iowa Hospital Neuroradiology Service. Clinical records and CT studies were reviewed to identify risk factors for ICH and determine the precise location of the hematomas. The vast majority of patients had unenhanced CT studies. Patients with head trauma, CT evidence of predominant subarachnoid hemorrhage, or prior histo-
ries of intracranial neoplasms, aneurysms, or arteriovenous malformations (AVMs) were excluded.

Patients were placed into one of three categories: 1) hypertensive, 2) non-hypertensive and those with 3) bleeding diathesis, (including those receiving anticoagulants). Hypertension was defined in adults when the average of two or more diastolic blood pressures (BPs) on at least two subsequent visits was 90 mm Hg or higher, or when the average of multiple systolic BPs on two or more subsequent visits was consistently greater than 140 mm Hg. 1 Electrocardiographic evidence of left ventricular hypertrophy or cardiomegaly by roentgenography supplemented by hypertensive changes on funduscopic examination, were considered signs of untreated hypertension. Young patients with a history of sympathomimetic drug abuse or pre-eclampsia were also included in the hypertensive group. Angiographic vessel displacement, indicative of mass effect, was not utilized as a criterion of a diagnostic related abnormality.

Results

One hundred and two patients (53 men and 49 women), ranging from 18 to 82 years of age, had 115 cerebral arteriographic studies. Forty-seven patients were hypertensive, 50 were non-hypertensive and 5 had a bleeding diathesis. The mean age was 57.8 years (range 21 to 82 years) for the hypertensive group, 46.7 years (range 18 to 82 years) for the non-hypertensive group, and 59.2 years (range 49 to 72 years) for those with bleeding diathesis.

Among all patients, 28 diagnostic arteriographic studies were identified (table 1). Cerebral arteriography was positive in 6 (12.8%) hypertensive patients (table 2), and in 22 (44%) non-hypertensive patients (table 3). No additional information was gained by repeated angiography, during hospitalization (7–21 days after first arteriogram), in 9 non-hypertensive patients (one patient had 3 studies), 2 hypertensive patients and 1 patient with bleeding diathesis. Two normotensive patients, ages 63 and 72 with biopsy proven amyloid angiopathy as the etiology for their lobar hemorrhages, had no diagnostic arteriographic studies. The yield of diagnostic arteriography according to ICH location among normotensive patients was as follows: 47% for lobar hemorrhages and 37.5% for non-lobar (table 4). The yield of arteriography among all hypertensive individuals, excluding the 3 patients with intraventricular bleeds, was 6.8% (table 2).

<table>
<thead>
<tr>
<th>TABLE 1 Angiographic Findings in 102 Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysm (n)</td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>47 hypertensive patients</td>
</tr>
<tr>
<td>50 nonhypertensive patients</td>
</tr>
<tr>
<td>5 patients with bleeding diathesis</td>
</tr>
</tbody>
</table>

*SSS = superior sagittal sinus thrombosis; *CC = choriocarcinoma.

Table 2

<table>
<thead>
<tr>
<th>CT location of ICH (n)</th>
<th>Aneurysm (n)</th>
<th>AVM (n)</th>
<th>Moya-moya (n)</th>
<th>Other (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lobar (23)</td>
<td>2</td>
<td>0</td>
<td>1 SSS*</td>
<td></td>
</tr>
<tr>
<td>Putamen (13)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Thalamus (4)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Cerebellum (3)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Medulla (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Intraventricular (3)</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(n = 47).

*2 SSS thrombosis, 1 choriocarcinoma.

Overall results: diagnostic arteriograms 6 (12.8%).

Excluding intraventricular bleeds, the yield would be 20/47 (42.6%).

Discussion

In 1961, Fisher reported two hypertensive patients with large basal ganglion hemorrhages in whom arteriography was normal. Nine years earlier, Scott noted that more than half of his 32 patients with spontaneous ICH had a history of hypertension and recommended arteriography only if there was no history of hypertension. With the advent of CT in the early 1970's, the role of cerebral arteriography for the evaluation of hypertensive hemorrhage has decreased. Scott and Miller reported 9 patients with small supratentorial ICH simulating cerebral infarction. ICH was diagnosed by CT; arteriography did not provide any additional information. Douglas and Haerer believed, when the diagnosis of hypertensive ICH was obvious, it was "ethically unacceptable" to perform arteriography. Other investigators have suggested that arteriography should be restricted to patients with ICH outside the basal ganglia.

In our experience, arteriography did not provide etiological information in hypertensive patients who had hemorrhages in the medulla, cerebellum, thalamus, or putamen. The diagnostic yield of arteriography is higher in hypertensive patients who had hemorrhages outside the basal ganglia. Arteriography was helpful in three hypertensive patients with isolated intraventricular hemorrhages.

Table 3

<table>
<thead>
<tr>
<th>CT location of ICH (n)</th>
<th>Aneurysm (n)</th>
<th>AVM (n)</th>
<th>Moya-moya (n)</th>
<th>Other (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lobar (34)</td>
<td>5</td>
<td>6</td>
<td>2</td>
<td>3*</td>
</tr>
<tr>
<td>Putamen (4)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Thalamus (2)</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Pons (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
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<tr>
<td>Caudate (1)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td></td>
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<tr>
<td>Midbrain (4)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Corpus callosum (1)</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Intraventricular (3)</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

(n = 50).

*2 SSS thrombosis, 1 choriocarcinoma.

Overall results: diagnostic arteriograms 22 (44%).

Excluding intraventricular bleeds, the yield would be 20/47 (42.6%).
TABLE 4  Lobar Hemorrhages in Nonhypertensive Patients

<table>
<thead>
<tr>
<th>CT location of ICH (n)</th>
<th>Aneurysm (n)</th>
<th>AVM (n)</th>
<th>Moyamoya (n)</th>
<th>Other (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal (11)</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Parietal (6)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Temporal (11)</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Occipital (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Biparietal (3)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 SS*</td>
</tr>
<tr>
<td>Multilobar (2)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>—</td>
</tr>
</tbody>
</table>

(n = 34).

*SSS = superior sagittal sinus thrombosis; *CC = choriocarcinoma.

Overall results: diagnostic arteriograms 16 (47%). Those without lobar hemorrhages (16 pts) had 6 diagnostic arteriograms (37.5%).

The results of our study confirm the value of cerebral arteriography among normotensive patients, especially those with lobar hematomas. Hung demonstrated a high yield of arteriography in patients with lobar hemorrhages; among his 41 normotensive patients, 32 (78%) had either an AVM or aneurysm.

All hypertensive or normotensive patients with putaminal hemorrhages had normal arteriograms in our study. We believe that patients in this category should not routinely undergo cerebral angiography, especially if the patient is elderly or has multiple medical problems.

More than half of the non-hypertensive patients did not have an etiology for the hematomas, as established by arteriography. There are several possible explanations. Arteriography may not detect "cryptic" vascular malformations. CT and arteriography may fail to identify a neoplasm presenting as an ICH. Cerebral amyloid angiopathy is now recognized as a frequent cause of ICH, particularly among older patients. In our study, arteriography was performed in 42 patients older than 60 years, 34 had nondiagnostic studies. Two of these had biopsy proven amyloid angiopathy. Amyloid angiopathy could explain ICH in several of our other elderly patients with negative angiograms. Normal arteriography does not eliminate amyloid angiopathy as a cause of ICH in the elderly. Finally, some investigators feel that ICH can occur spontaneously without an explanation.

In conclusion, our data suggest cerebral arteriography provides valuable etiological information in non-hypertensive patients with ICH. Normotensive individuals with lobar hemorrhages are the most likely to have diagnostic angiographic studies. Hypertensive patients with isolated ventricular bleeding should have arteriography. The yield from arteriography is low in hypertensive patients with intraparenchymal hematomas. Arteriography is probably not indicated in hypertensive or normotensive patients with putaminal hemorrhages.

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References

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