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CT Angiography of Cerebral Vasospasm with Conventional Angiographic Comparison

Robert P. Ochi, Pedro T. Vieco, and Cordell E. Gross

Summary: Two patients with subarachnoid hemorrhage underwent CT angiography and conventional angiography at presentation. In each patient, both studies were repeated after the onset of intracranial vasospasm. In both cases, CT angiograms were able to demonstrate convincingly the conventional angiographic findings. CT angiography may prove useful in the evaluation of vasospasm in patients with subarachnoid hemorrhage.

Index terms: Computed tomography, three-dimensional; Vasospasm

An estimated 28 000 ruptured intracranial aneurysms occur each year in North America; approximately 8500 of these patients survive to receive treatment (1). Among patients with intracranial aneurysms, roughly 2% per year will experience subarachnoid hemorrhage resulting from aneurysmal rupture (2). The most debilitating complication of aneurysmal subarachnoid hemorrhage is cerebral vasospasm, accounting for 14% of deaths or severe disability in those treated (3).

Conventional angiography is one method of diagnosing this complication. However, the risk of performing this procedure in the critically ill patient can limit its application. Transcranial Doppler sonography is noninvasive and rapidly performed, but does not provide anatomic information and is limited to a small acoustic window (4).

Computed tomographic (CT) angiography has recently been used to evaluate the vessels in the region of the circle of Willis (5–7). This technique offers the potential for a rapid, minimally invasive means of accurately diagnosing and monitoring this complication. We report our findings in two patients with clinical evidence of cerebral vasospasm shown by both CT angiography and conventional angiography.

Case Reports

Case 1

A 50-year-old man with a history of hypertension presented with acute onset of a severe frontal headache. CT scans showed subarachnoid hemorrhage involving the suprasellar and ambient cisterns, Hunt-Hess class I. A CT angiogram and a conventional angiogram (Fig 1A and B) did not reveal the source of hemorrhage. Eleven days after admission, clinical signs and symptoms of cerebral vasospasm were noted. Findings at transcranial Doppler sonography, performed just prior to angiography, were interpreted as normal. However, vasospasm involving the basilar artery, the posterior cerebral arteries bilaterally, and the left M1 segment of the middle cerebral artery, was identified at catheter angiography. The P1 and P2 segments of the posterior cerebral arteries (Fig 1C) were most severely affected. Findings at CT angiography performed 2 days later (Fig 1D) were concordant with the above findings. Vasospasm was successfully treated with volume expansion. The patient was ultimately discharged neurologically intact; the source of his subarachnoid hemorrhage was never identified.

Case 2

A 40-year-old woman presented with sudden onset of a posterior occipital headache. CT examination and lumbar puncture were confirmatory for subarachnoid hemorrhage, Hunt-Hess class I. CT angiography and conventional angiography (Fig 2A and B) were performed. A left posteroinferior cerebellar artery aneurysm was identified at conventional angiography; surgical repair was performed without incident. On the 10th postoperative day, severe vasospasm of the basilar artery, P1 segments of the posterior cerebral arteries bilaterally, right M1 segment of the middle cerebral artery, and supraclinoid portion of the left internal carotid artery was shown by conventional angiography (Fig 2C). The remainder of the vessels studied were unremarkable. CT angiography repeated 2 days later showed similar findings (Fig 2D). Of interest, findings at serial transcranial Doppler sonography performed concor-

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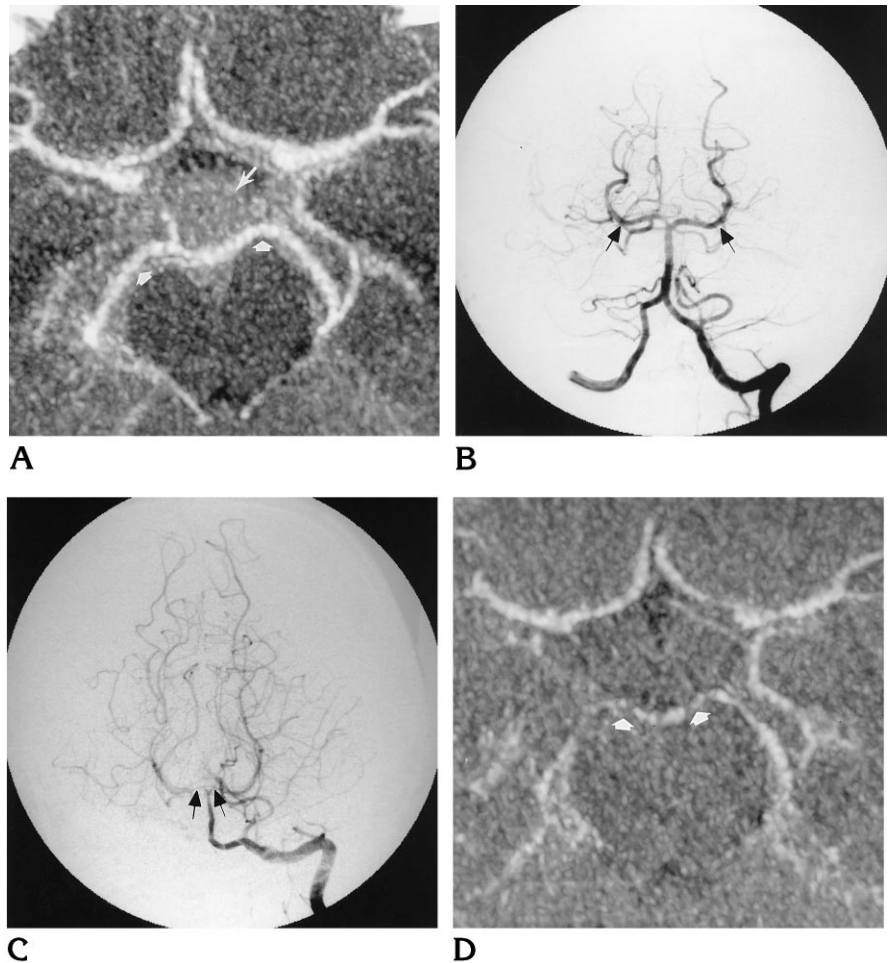
Fig 1. Case 1: 50-year-old man with subarachnoid hemorrhage followed by vasospasm.

A, CT angiogram (MIP display) obtained at presentation shows normal vessels of the circle of Willis. Note the normal caliber of the posterior cerebral arteries bilaterally (*short arrows*) and the presence of recent subarachnoid hemorrhage within the suprasellar cistern (*long arrow*).

B, Conventional digital subtraction angiogram, left vertebral injection, posteroanterior view. The basilar artery and posterior cerebral arteries (*arrows*) are normal.

C, Conventional digital subtraction angiogram, left vertebral injection, posteroanterior view, obtained after onset of vasospasm, shows the spastic appearance of the basilar artery and P1 segments bilaterally (*arrows*).

D, CT angiogram obtained after onset of vasospasm shows a reduction in the caliber of many segments compared with the prior study, in particular the P1 segments bilaterally (*arrows*).



dantly during this period were interpreted as normal. The patient's vasospasm was treated effectively with volume expansion, hemodilution, and inotropic support.

Both patients were studied with CT angiography and digital subtraction angiography. Each was evaluated at presentation and later at the clinical onset of vasospasm. CT angiography was performed using a Hi-speed helical scanner (General Electric Medical Systems, Milwaukee, Wis). Imaging parameters included 120 kV, 280 mA, 1 second, 1-mm collimation, 1:1 pitch, with a 12.8-cm field of view. Sixty axial source images were obtained from the foramen magnum through the circle of Willis after intravenous administration of contrast material (Omnipaque 300, Sanofi-Winthrop, Barcelona, Puerto Rico), delivered via an antecubital vein at 2 mL/s for a total of 100 mL. Acquisition of source images was started 30 seconds after the start of contrast injection. Images were transferred to a GE Advantage Windows Workstation (General Electric Medical Systems). Each data set was then reconstructed using a maximum-intensity-projection (MIP) algorithm. Image reconstruction and interpretation were performed by a neuroradiologist. Conventional digital subtraction angiography was performed via femoral catheterization using a 0.3-mm focal spot and a 1024 × 1024 matrix (Model

DFP-60A, Toshiba Corp., Kawasaki, Japan). Selective bilateral carotid and vertebral injections were performed in the anteroposterior and lateral projections. Magnetic resonance (MR) angiography was not performed in either patient.

Discussion

Cerebral vasospasm is the leading cause of morbidity and mortality in patients with aneurysmal subarachnoid hemorrhage (1, 3). Time of onset ranges from 4 to 9 days after hemorrhage, with a peak at the seventh day (3). From 40% to 70% of affected patients have some reduction in caliber of one or more of the arteries of the circle of Willis (8). The diagnosis is often based on clinical findings and the exclusion of other complications such as recurrent hemorrhage, hematoma, hydrocephalus, or metabolic derangement. Correct diagnosis of vasospasm is useful before embarking on complicated, potentially hazardous therapy (3). At our institu-

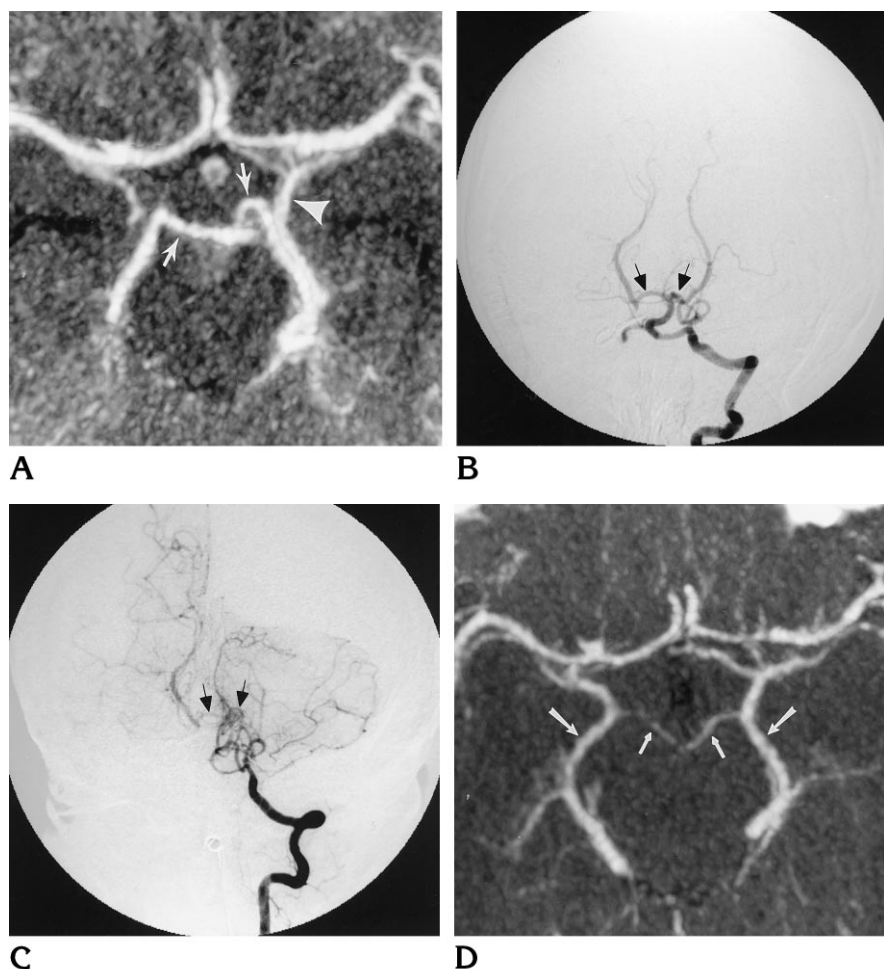


Fig 2. Case 2: 40-year-old woman with subarachnoid hemorrhage followed by vasospasm.

A, CT angiogram (MIP display) obtained at presentation shows normal vessels of the circle of Willis. Note the normal caliber of the P1 segments of the posterior cerebral arteries bilaterally (arrows) and the basal vein of Rosenthal (arrowhead).

B, Conventional digital subtraction angiogram, left vertebral injection, posteroanterior view. The basilar artery and P1 segments of the posterior cerebral arteries (arrows) are normal.

C, Conventional digital subtraction angiogram, left vertebral injection, posteroanterior view, obtained after onset of vasospasm, shows the spastic appearance of the basilar artery and P1 segments bilaterally (arrows).

D, CT angiogram obtained after onset of vasospasm shows the P1 segments are severely narrowed bilaterally (short arrows). Note also the basal veins of Rosenthal (long arrows).

tion, treatment of these patients generally depends on the severity of vasospasm encountered. Severely affected patients are treated with hemodilution, induced hypertension, and hypervolemia. Less ill patients are treated by intravascular volume-expanding agents. Diagnosis is made clinically and confirmed predominantly by transcranial Doppler sonography. Catheter angiography is reserved for patients in whom the clinical and sonographic findings are discordant.

The radiologic diagnosis of this phenomenon is typically made by conventional angiography, a procedure that is not without risks. Central nervous system complications from this procedure are reported to be as high as 0.9% in healthy persons (9). Additionally, less serious but more common complications of angiography such as nausea, vomiting, puncture site hematoma, hypotension, arrhythmia, and heart failure may have more clinical significance in the critically ill patient.

CT angiography has recently been applied in the evaluation of the circle of Willis (5-7, 10, 11), mostly for the detection of berry aneurysms. CT angiography offers the ability to show three-dimensional detail of the intracranial vasculature in a rapid, easily reproducible fashion, and has proved useful in screening for intracranial aneurysms (5, 10), evaluating acute subarachnoid hemorrhage (6), planning surgical therapy, and assessing aneurysmal clip placement (7).

Disadvantages of CT angiography include radiation exposure and contrast administration. Moreover, the images are of poorer resolution than those obtained by conventional angiography. Vessel anatomy adjacent to bone or calcium density, such as at the skull base, may be difficult to evaluate (6). Venous opacification may also impair evaluation of arterial anatomy; the cavernous portion of the internal carotid artery being one such example (6). Beam-hardening artifacts caused by dental amalgam or

other metallic objects can also degrade image quality. Postprocessing requires a finite amount of physician or technologist time to provide the most useful images, and, as with any technique, there will be interobserver variability in both image preparation and interpretation. The interpreter must be familiar with the variant arterial and venous anatomy of the region of the circle of Willis to avoid misdiagnosis. For example, the inexperienced observer could mistake the basal veins of Rosenthal (Fig 2C) for the posterior cerebral arteries. In our two patients, MIP reconstructions were used instead of shaded surface displays. While there are advantages to each of these techniques (12), we chose to use the MIP images because of the diminished caliber of the vasospastic vessels. Shaded surface displays may inadvertently exclude small vessels of the circle of Willis (12).

MR angiography has been used in the diagnosis of intracranial aneurysms (10, 13–16), and it shares the advantage of multiplanar postprocessing capabilities of CT angiography. Reported sensitivities in diagnosing intracranial aneurysms range from 67% to 95%, with a spatial resolution comparable to that of CT angiography (13–15). Other advantages include lack of exposure to ionizing radiation and no requirement for intravenous contrast administration. However, for obtunded, often mechanically ventilated, critically ill patients, MR imaging may not be ideal owing to relatively prolonged acquisition times and limited patient access. Electric and metallic devices, such as aneurysmal clips, also preclude its routine use in the postoperative patient (17). To our knowledge, MR angiography has not been shown to be useful in the examination of patients with vasospasm after subarachnoid hemorrhage.

Transcranial Doppler sonography is often used to monitor patients during the posthemorrhagic period (18). Advantages of this technique include its rapidity and noninvasiveness, allowing frequent monitoring of the intracranial circulation. This technique, however, is limited by the relatively small area of intracranial vasculature that is sonographically accessible (4). Additionally, it requires training and experience to perform and is inherently operator-dependent. Moreover, the velocities reported must be considered in clinical context. Relying purely on the assumption that lower velocities reflect wider vessel caliber ignores the possibility that elevated intracranial pressure limits the cerebral

perfusion pressure, and hence the measured velocity (19). In these patients, the possibility exists for falsely excluding vasospasm in a moribund patient with elevated intracranial pressure. Transcranial Doppler sonography may be useful for frequent monitoring, but it does not offer the anatomic detail of CT angiography.

CT angiography provides several advantages over MR angiography for evaluating vasospasm. The acquisition of data (60 seconds) is much faster than with MR angiography at present (6). Motion artifacts are also reduced, and the critically ill patient is not left relatively isolated from support staff for as long a time. The vascular anatomy shown corresponds to opacification by intravenous contrast material, and is independent of slow or complex flow. The flow-dependent imaging characteristics inherent to MR angiography may theoretically limit the evaluation of vasospasm.

In summary, CT angiography shows promise as a rapid, minimally invasive method of evaluating vasospasm of the intracranial circulation. While the use of CT angiography did not alter the management of the patients reported here, this technique may prove to be a suitable alternative to catheter angiography for the diagnosis and treatment of patients with intracranial vasospasm.

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