The Striate Artery, Hematoma, and Spot Sign on Coronal Images of Computed Tomography Angiography in Putaminal Intracerebral Hemorrhage

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**Background and Purpose**—A spot sign is a bright spot on computed tomography angiography source images, which is predictive of hematoma growth in spontaneous intracerebral hemorrhage, although the cause of the spot sign is unclear. Our aim was to investigate the spot sign seen on computed tomography angiography and a striate artery, which is a presumed site of intracerebral hemorrhage bleeding origin in the putamen.

**Methods**—In consecutive cases of spontaneous intracerebral hemorrhage in the putamen, spot signs and striate arteries were evaluated. Coronal reformat images of computed tomography angiography were created to visualize the striate arteries. Acute deterioration, defined as hematoma enlargement, emergency hematoma removal, or death within the day of admission, was reviewed.

**Results**—Of the 141 patients undergoing computed tomography angiography, 15 of the 30 patients (50%) who had spot signs showed an intrahematoma striate artery (termed spot and tail sign), which was a linear density extending from the middle cerebral artery toward the spot sign. Acute deterioration occurred more frequently in patients who had a spot and tail sign compared with patients who had spot signs without intrahematoma striate arteries (P<0.05). Multivariate analysis revealed that hematoma volume, spot signs, and intrahematoma striate arteries were independent predictors of acute deterioration (P<0.05).

**Conclusions**—The presence of a spot and tail sign, assumed to indicate active bleeding from the striate artery, could be a more sensitive predictor of acute deterioration than the presence of a simple spot sign. (Stroke. 2013;44:1830-1832.)

Key Words: computed tomography angiography ■ intracerebral hemorrhage ■ perforator ■ predictors ■ spot sign

A spot sign seen in computed tomography angiography (CTA) has recently been shown to predict hematoma expansion in patients presenting acutely with spontaneous intracerebral hemorrhage (ICH). Although a spot sign is generally assumed to reflect continued bleeding from a ruptured vessel, little is known about the cause of a spot sign. A spontaneous ICH located in the putamen, which is the most frequent site of a spontaneous ICH, is caused by the rupture of striate arteries based on pathological investigation.4,5 Coronal reformat images of CTA source images can make striate arteries visible because the striate artery courses parallel to the coronal image plane. In the present study, we investigated the relationship among the hematoma, the striate arteries, and the spot signs in putaminal ICH on coronal images of CTA.

Methods

We retrospectively assessed consecutive patients diagnosed with spontaneous ICH that was located mainly in the putamen between January 2009 and January 2013. Patients presenting with hemorrhagic stroke admitted to our neurosurgical unit routinely undergo their first noncontrast computed tomography (CT) scan on arrival, followed by a CTA study. Coronal multiplanar reformat images of CTA were created as 3.0-mm-thick images to observe the striate arteries originating from the M1 segment of the middle cerebral artery.

A spot sign is defined as a contrast density on CTA visible within the hematoma margin.2,3 A striate artery is defined as a linear contrast enhancement originating from the M1 on coronal images of CTA. Hematoma enlargement is defined as a volume increase of ≥12.5 mL or ≥33%. A follow-up CT scan for evaluation of hematoma expansion was frequently not performed in patients who were undergoing emergency hematoma removal and in patients who had died within the day of admission. Acute deterioration, defined as hematoma enlargement on follow-up CT, emergency hematoma removal within the day of admission, or death within the admission day, was reviewed (see detailed patient information, image acquisition/analysis, and statistical methods in the online-only Data Supplement).

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Results
We treated 238 consecutive patients diagnosed with spontaneous putaminal ICH. Three patients admitted later than 24 hours after onset and 94 who failed to undergo a CTA study at admission were excluded. A total of 141 patients met the inclusion criteria, with a mean age of 64.3±13.1 years (range, 34–92 years). Mean initial ICH volume was 54.0±47.8 mL (range, 3–236 mL). Emergency hematoma removal was performed within the day of admission in 44 patients, including 8 undergoing the second CT within the day of admission before the surgery. Fourteen patients died by the following day of admission. In 91 patients undergoing follow-up CT, 9 patients showed hematoma enlargement, including 2 patients undergoing emergency hematoma removal. Therefore, 65 patients (46.1%) belonged to an acute deterioration group. The remaining 76 patients (53.9%) did not show acute deterioration.

In 133 patients (94.3%), striate arteries from the M1 segment were observed on coronal images of CTA. Medial, anterior, lateral, and posterior displacements of the striate arteries were observed in 88 (62.4%), 18 (12.8%), 3 (2.1%), and 1 (0.7%) patients, respectively (Figure A). The striate arteries running within a hematoma were shown in 20 patients (14.2%).

The spot signs were present in 30 patients (21.3%). The Table shows the relationships among spot signs, intrahematoma striate arteries, acute deterioration, and clinical outcome. The number of patients who had acute deterioration was significantly larger in patients with spot signs compared with patients who did not have spot signs (P<0.0001). In 15 of the 30 patients (50%) who had spot signs, an intrahematoma striate artery, which was a linear density extending from the M1 into the hematoma and coursing through it toward the spot sign, was observed on coronal images of CTA (Figure B). All the 15 patients (100%) who had both spot signs and intrahematoma striate arteries showed acute deterioration. Nine of the 15 patients (60.0%) who had spot signs but no intrahematoma striate arteries developed acute deterioration. The number of patients who developed acute deterioration was significantly larger in patients who had both spot signs and intrahematoma striate arteries in comparison with patients who had spot signs but no intrahematoma striate arteries (P=0.0169). In 5 patients who did not have spot signs, intrahematoma striate arteries were observed on CTA coronal images (Figure C). In 71 patients who were admitted within 3 hours and undertook follow-up CT, hematoma enlargement on the follow-up CT was observed in 3 of 3 patients (100%) with spot signs and intrahematoma striate arteries, in 2 of 8 (25.0%) with simple spot signs, and in 3 of 60 (5.0%) without spot sign.

The multivariate analysis, including patient age, sex, time from onset to emergency department presentation, Glasgow Coma Scale score at admission, systolic blood pressure at admission, use of antiplatelets and anticoagulants, hematoma volume, intraventricular hemorrhage, spot signs, and intrahematoma striate arteries, revealed that 3 independent factors are correlated with acute deterioration: hematoma volume (P<0.0001; odds ratio, 1.071 [1.049–1.101]); spot signs (P=0.0038; odds ratio, 10.747 [2.148–60.261]); and intrahematoma striate arteries (P=0.0034; odds ratio, 26.127 [2.672–653.862]).

Discussion
We termed a combination of the spot sign and the intrahematoma striate artery spot and tail sign because of the distinctive appearance of the combination on CTA coronal images (Figure B). On an angiogram of spontaneous ICH, extravasation of contrast medium from a striate artery was reported.6 We presumed that the spot and tail sign on CTA coronal images was equivalent to the extravasation from a striate artery on an angiogram. In our study, acute deterioration occurred in ICH that had spot and tail sign more frequently than those that had spot sign but no intrahematoma striate arteries. In cases of spot and tail sign, continuous blood supply through the striate artery to the rupture point, which was expressed as a liner enhancement on CTA, was considered to facilitate hematoma expansion. In cases of spot signs without intrahematoma striate artery, a tortuous course and stagnant blood flow of a ruptured striate artery were assumed to make the striate artery invisible on CTA. Bleeding from veins or damaged perihematoma tissue5,7 might be another reason for the absence of a visible striate artery.

Figure. Striate arteries on coronal images of computed tomography angiography (CTA). A. Striate arteries (arrows) displaced medially by a hematoma. B. A striate artery (arrows) originating from the M1 segment of the middle cerebral artery and coursing into a hematoma to terminate as a spot sign (arrow head). Demonstration of both the spot sign and the striate artery on coronal images of CTA is termed spot and tail sign. C. A striate artery (arrows) coursing within a hematoma that had no spot sign.
In conclusion, a spot and tail sign, assumed to indicate active bleeding from the striate artery, could be a more sensitive predictor of acute deterioration than a simple spot sign.

Disclosures
None.

References
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SUPPLEMENTAL MATERIAL

The striate artery, hematoma, and spot sign on coronal images of CT angiography in putaminal intracerebral hemorrhage

Supplemental Methods

Patient population

We retrospectively assessed consecutive patients diagnosed with spontaneous intracerebral hemorrhage (ICH) that was located mainly in the putamen between January 2009 and January 2013. Patients admitted later than 24 hours after onset of ICH were excluded. Patients who failed to undergo a computed tomography angiography (CTA) study at admission were also excluded. This series of patients did not include those that had a hemorrhage caused by intracranial aneurysm, arteriovenous malformation, moyamoya disease, and cavernous hemangioma. All patients with neurological symptoms were evaluated by neurosurgeons. Patients presenting with hemorrhagic stroke admitted to our neurosurgical unit routinely undergo their first noncontrast CT scan on arrival followed by a CTA study. Our institutional review board approved a retrospective study of patients with ICH admitted to our institution.

Image Acquisition

CT imaging was performed on either a 40-slice (2009–2011) or a 128-slice (2012–2013) CT (Brilliance; Philips, Best, The Netherlands, and Definition AS; Siemens, Erlangen, Germany) scanner. Noncontrast head imaging was acquired from the skull base to vertex with parameters: 120 kVp; 360 mA; 1 second/rotation. Axial reformats were 5 mm for noncontrast CT. CTA studies were acquired from C4 to the vertex in the helical mode with the following parameters: 4 mL/sec contrast medium through an antecubital vein via at least an 18- or 20-gauge angiocatheter; 10 second delay; 100 kVp; 230 mA; 0.5 second/rotation; 0.6 mm slice thickness, table speed 0.55 mm/rotation. Postprocessing, including all multiplanar reformats, was performed by the CT technologists at the CT operator’s console. Coronal multiplanar reformat images were created as 3.0-mm-thick images to observe the striate arteries originating from the
M1 of the middle cerebral artery (MCA). All images were viewed on PACS workstation.

**Imaging Analysis/Interpretation**

All studies were evaluated by a single neurosurgeon who was blinded to clinical and radiological outcome for the presence or absence of spot sign and striate artery by simultaneously visualizing noncontrast CT studies cross-linked with coronal axial CTA reformats. An identical window was applied to all studies that best visualized vessel density (Approximately WW 200/80). A CTA spot sign was defined as a contrast density on CTA visible within the hematoma margin.\textsuperscript{1-3} A striate artery is defined as a linear contrast enhancement originating from the M1 portion of the MCA on coronal images of CTA. Hematoma volume at presentation and during follow-up study was calculated by separate neurosurgeons by measuring baseline volume and follow-up noncontrast CT hematoma volume using the previously validated ABC/2 method.\textsuperscript{4} Hematoma enlargement was defined as a volume increase of ≥12.5 mL or 33% or more.\textsuperscript{2,3,5}

**Data collection**

Clinical data were obtained through chart review. The following data were recorded: patient age and sex, level of consciousness (Glasgow coma scale [GCS] score) at admission, systolic blood pressure (BP) at admission, use of antiplatelet agents, use of anticoagulants, and time from ICH onset to admission. Clinical outcomes were assessed by the modified Rankin Scale (mRS) score at 30 days or at discharge if that were sooner. Poor outcome was defined by the following: (1) the inability to walk or attend to own bodily needs without assistance (mRS 4), (2) being bedridden, incontinent, and requiring constant nursing care and attention (mRS 5), or (3) death after hospital discharge (mRS 6).\textsuperscript{6}

Immediately after diagnosis of ICH, systolic BP was maintained below 150 mm Hg by continuous intravenous administration of nicardipine for 24 hours. Emergency hematoma removal was performed in patients less than 80 years old with severe consciousness disturbance by a mass effect of the hematoma or with impending tentorial herniation by the hematoma. A follow-up CT scan for evaluation of hematoma expansion frequently failed to be performed in patients who were undergoing
emergency hematoma removal and in patients who had died within the day of admission. Acute deterioration was defined as hematoma enlargement on follow-up CT, emergency hematoma removal within the day of admission, or death within the admission day. If only the hematoma enlargement on follow-up CT had been used as the indicator, other cases of hematoma enlargement without confirmation by the second CT, included in the emergency hematoma removal and death by the following day, might have been missed. Patients who undertook emergency hematoma removal or who were dead by the following day possibly included some patients who had no change of hematoma volume after the first CT.

**Statistical Analysis**

Univariate analysis was performed using chi-squared analysis and Fisher’s exact probability test for categorical variables. Numerical data were expressed as the mean ± SD.

Each clinical variable was analyzed using the multivariate logistic regression analyses to find possible significant predictors under evaluation, which was reduced by successively removing the least significant variable from the model. All variables that had a $p < 0.10$ were kept in the final model. Analyses resulting in $p$ values of less than 0.05 were considered statistically significant. All statistical analyses were performed by use of JMP 10 (SAS Institute Inc. Cary, NC, USA).

**Supplemental References**


