CASE REPORT

A Hyperdense Artery Sign and Middle Cerebral Artery Dissection

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Abstract

We describe a rare case of spontaneous middle cerebral artery (MCA) dissection that caused cerebral infarction and subarachnoid hemorrhage (SAH), which also presented with a hyperdense artery sign. A hyperdense artery sign of the MCA in acute cerebral infarction strongly indicates thromboembolic MCA occlusion, which is often treated with thrombolytic therapy. However, thrombolytic therapy for intracranial artery dissections has both risks and benefits, due to the association of artery dissections with SAH. Therefore, it is important to keep in mind that an MCA dissection can also cause cerebral infarction with a hyperdense artery sign, particularly in young patients presenting with headache.

Key words: Hyperdense middle cerebral artery sign, Computed tomography, Intracranial artery dissection, Cerebral infarction

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Introduction

A hyperdense artery sign of the middle cerebral artery (MCA) in acute cerebral infarction strongly indicates thromboembolic MCA occlusion (1), which is often treated with thrombolytic therapy (2). However, dissection of the intracranial cerebral arteries can cause a subarachnoid hemorrhage (SAH), as well as cerebral infarction (3); therefore, thrombolytic therapy is generally not indicated in such cases. We describe a case of spontaneous MCA dissection that caused cerebral infarction and SAH, which also presented with a hyperdense artery sign. We also discuss the literature relating to intracranial artery dissection associated with a hyperdense artery sign.

Case Report

A 40-year-old man with hypertension was admitted due to the sudden onset of disturbance of consciousness without any traumatic injury. His pulse was regular (90/min), and his blood pressure was 162/126 mmHg. Neurological examination revealed drowsiness (Japan Coma Scale II-10), motor aphasia, and right-sided hemiplegia. National Institutes of Health Stroke Scale (NIHSS) was 15. Laboratory examinations were normal, including α1-antitrypsin level and hematocrit (45.9%).

Brain computed tomography (CT), performed on admission (Day 1), showed a hyperdensity at the M1 segment through to the posterior trunk of the M2 segment of the left middle cerebral artery (MCA) (Fig. 1a) and hypodensity of more than one-third of the left MCA territory. The MCA attenuation value was 66 Hounsfield units (HU) on the left and 39 HU on the right (the attenuation ratio of the affected vessel to the normal vessel was 1.69). Brain diffusion-weighted imaging (DWI) and time-of-flight magnetic resonance angiography (TOF MRA) were performed the same day and immediately followed by brain CT. DWI showed that the acute infarction was restricted to the left MCA area. TOF MRA showed slight dilatation at the distal M1 segment, but patency of the left MCA (Fig. 1b). No embolic sources were detected by either transesophageal echocar-
Figure 1. a. An unenhanced brain CT scan, performed on Day 1, shows increased attenuation at the M1 segment of the left middle cerebral artery (arrow). The MCA attenuation value is 66 Hounsfield units (HU) on the left and 39 HU on the right. b. Time-of-flight MR angiography (Advantage 1.5 T system, TR 57 ms, TE 4.5 ms, Flip angle 30°, section thickness 0.9 mm), performed on Day 1 and immediately followed by brain CT, shows patency of the left MCA.

Figure 2. A brain CT, performed on Day 2, demonstrates SAH (arrowheads) and hypodensity with edema in the left MCA area.

Figure 3. Images of selective left internal carotid angiography done with a rotational three-dimensional digital subtraction angiography system. a. Initial examination performed on Day 11 shows an aneurysmal dilatation at the distal M1 segment (arrow) and a “pearl-and-string sign” at the M1 segment through to the posterior trunk of the M2 segment (arrowheads). b. Follow-up examination performed on Day 25 shows reduction of the aneurysmal dilatation (arrow) and improvement of the “pearl-and-string sign” (arrowheads). A narrowing of the M1 segment of the MCA is also seen.

diography or conventional carotid ultrasonography.

On Day 2, brain CT demonstrated SAH and hypodensity with edema in the left MCA area (Fig. 2). On Day 4, the patient’s disturbance of consciousness gradually improved, but he began to complain of a left-sided headache, which gradually worsened without a fever developing. On the same day, brain CT showed that the hyperdensity of the left MCA was still present (the MCA attenuation value was 63 HU on the left and 40 HU on the right, while the attenuation ratio was 1.58). Meanwhile, TOF MRA, which was performed immediately after brain CT, demonstrated patency and aneurysmal dilatation at the posterior trunk of the left MCA. On Day 7, xanthochromia was found on examination of the cerebrospinal fluid. Selective left internal carotid angiography using a rotational three-dimensional digital subtraction angiography system (3-D DSA) was conducted on Day 11 and showed an aneurysmal dilatation at the distal M1 segment and a “pearl-and-string sign” at the M1 segment through to the posterior trunk of the M2 segment (Fig. 3a). On follow-up CT on Day 14, the hyperdensity of the left MCA was no longer seen, and the MCA attenuation value was 41 HU on the left and 40 HU on the right (the attenuation ratio was 1.03). On Day 20, 3-dimensional spoiled gradient-recalled acquisition (SPGR) identified a narrowing of the proximal region of the M1 segment of the left MCA (Fig. 4a). The distal M1 segment of the left MCA was more
distended than that of the right MCA, and a laminar low intensity signal was seen along the rostral left MCA. These SPGR findings suggested the presence of a false lumen (Fig. 4b). T1-weighted images demonstrated a high intensity signal along the arterial wall at the M1 segment through to the posterior trunk of the M2 segment of the left MCA (arrowheads), thus suggesting an intramural hematoma. d. T1-weighted images with contrast show enhancement of the posterior trunk of the M2 segment of the left MCA (arrow), suggesting slight flow in the false lumen.

Discussion

Although a hyperdense artery sign can be seen in patients with a high hematocrit level or with calcification of the intracranial arteries (4), it is usually seen in patients with thromboembolic arterial occlusion, which results in an increased vessel attenuation value (1). Under such conditions, the MCA, which presents with a hyperdense artery sign, has a higher attenuation value than the contralateral MCA (5), which disappears within a couple of weeks (6). Both of these findings were demonstrated in our patient. Koo et al reported that a hyperdense artery sign associated with acute ischemic stroke can be distinguished from normal vessels and false positives by measuring the absolute attenuation and the ratios of the affected and the normal vessels; an absolute density of >43 HU and an MCA ratio of >1.2 define hyperdensity (7). In the present patient, the absolute densities of the affected MCA and the MCA ratios, which were obtained from brain CTs on Day 1 and Day 4, also met these requirements. Thus, there was a high degree of certainty regarding the presence of a hyperdense artery sign of the left MCA in our patient.

Some patients with major hemispheric stroke syndrome show a rapid recovery, also known as the “spectacular shrinking deficit” (SSD), due to migration of an embolus (8). The present patient had contradictory findings in that we could repeatedly demonstrate a hyperdense artery sign of the MCA on brain CTs and patency of the affected MCA on TOF MRAs (both done on Day 1 and Day 4). A previous report demonstrated that a hyperdense artery sign disappeared after the recanalization of the affected artery (6). The findings in the present patient could possibly be explained by the repeated occurrence of thromboembolic MCA occlusions and the migration of emboli. However, the patient’s uneventful clinical course from Day 1 to Day 4 conflicts with both repeated thromboembolic MCA occlusions and SSDs. Our case had some clinical features suggestive of an intracranial artery dissection (9), including: sudden onset...
cerebral infarction in a young person; a severe headache localized ipsilateral to the affected MCA; and demonstrated SAH. In addition to the clinical findings, the antemortem diagnosis of an intracranial artery dissection is based on the presence of certain radiological features. The pearl-and-string sign is known to be a reliable angiographic finding of an arterial dissection (9). An intramural hematoma on T1-weighted images and a double lumen on SPGR are MR findings that are suggestive of an arterial dissection (10). Furthermore, our patient showed serial spontaneous changes of affected vessel form on angiography, which is rare with atherosclerotic disease (9). Thus, both the clinical and radiological features in our case are consistent with an intracranial artery dissection.

Previously, five cases of intracranial artery dissection with a hyperdense artery sign on CT have been reported (11-13). Of these, one had an MCA dissection associated with a traumatic injury (11), one had an internal carotid artery-MCA dissection of unknown etiology (12), and three had vertebral artery dissections (in two patients these were related to exercise, in the other, etiology was unknown) (12, 13). Angiographic evaluations, which were performed in 4 patients, revealed 3 patients with distal arterial occlusion (12, 13) and one with irregular stenosis (12). Thus, including the current case, 2 cases of intracranial arterial dissection have demonstrated hyperdense artery signs without arterial occlusion. So how should the hyperdense artery sign be interpreted without arterial occlusion? It is known that plain CT in acute aortic dissection shows an intramural hematoma with crescentic high-attenuation areas along the aortic wall (14). Therefore, we considered that the hyperdense artery sign seen in the present patient might have been due to an intramural hematoma in the false lumen of the dissected MCA.

Nontraumatic dissection of the intracranial carotid circulation usually occurs in young persons and can cause cerebral infarction and SAH (3). There is no established standard medical treatment for dissections causing cerebral infarction. An acute ischemic stroke with HMCAS and without hypodensities of more than one-third of the MCA territory on baseline CT is considered to be an indication for intravascular t-PA administration (2). However, thrombolytic therapy for intracranial artery dissections has both risks and benefits, due to their association with SAH (15). Therefore, it is important to keep in mind that an MCA dissection can also cause cerebral infarction with a hyperdense artery sign, particularly in young patients presenting with headache.

References


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