### CT Visualization of Intracranial Arterial Thromboembolism

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**SUMMARY** Eight cases of presumed acute occlusion of intracranial arteries, 7 middle cerebral and 1 posterior cerebral, demonstrated the occluded artery as an abnormal high density structure on non-enhanced computed tomography. Three cases underwent subsequent angiography which confirmed the diagnosis. CT scanning is therefore able to visualize acute thromboembolism in the intracranial arteries. In the appropriate clinical setting of acute stroke, an artery visualized on non-contrast CT scan as diffuse high density and higher in density than other visualized vessels should be suspected as acutely occluded by clot.

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THE DIAGNOSIS of acute thrombotic or embolic processes in major arteries at the base of the brain may have therapeutic significance. In many cases of acute cerebral infarction, angiography is considered to be not indicated and commonly is deferred until there is clinical improvement. The ability to suggest the presence of major intracranial arterial occlusion on initial CT scan therefore is of clinical importance.

Demonstration of intravascular clots on CT scan is well known in large aneurysms and in venous sinuses.1-4 Yock5 demonstrated the possibility of CT diagnosis of chronic calcified emboli. No reports of the visualization of acute arterial thrombotic or thromboembolic occlusions are available. This report concerns eight instances of this phenomenon.

**Patients and Methods**

During a prospective study for evaluating the effectiveness of clinical diagnosis of ischemic cerebral disease6 one of the patients was found to have a high density visualization of the middle cerebral artery on the non-contrast scan. Angiography proved that the finding represented occlusion presumed to be due to thrombosis of the artery. A subsequent retrospective review of the CT scans of 735 patients with cerebral ischemia disclosed seven other cases including one of the posterior cerebral artery. Three of the eight patients were submitted to angiography confirming obstruction of the artery.

**Case 1**

A 41 year old right-handed physician presented at another hospital with a four day history of left-sided headache followed by sudden onset of aphasia and weakness of his right upper extremity. Five hours after onset of symptoms angiography revealed a left internal carotid occlusion (possibly due to dissection) and an unsuccessful attempt was made to reopen the artery. The patient suffered a series of epileptic seizures after surgery.

The patient was transferred to our institution the following day. Neurological examination revealed a left Horner’s syndrome, right homonymous hemianopia, complete right hemiplegia and total aphasia.

Computed tomography revealed a large area of low density in the territory of the left middle cerebral artery. The left middle cerebral artery was visualized on the non-contrast scan as a high density structure and was suspected to contain thrombus (fig. 1A). Subsequent angiography revealed abrupt occlusion of the origin of the middle cerebral artery with a rounded edge suggesting a middle cerebral artery embolus (fig. 1B and 1C).

The patient was treated with Dexamethazone and volume expanders. He remained densely aphasic and hemiplegic during his five days stay at this hospital.

**Case 2**

A 43 year old man with a history of migraine beginning in adolescence consulted an osteopath for neck
manipulation because of headache the day prior to admission to this institution. Two hours later he experienced a sudden onset of left-sided weakness. He exhibited inappropriate behaviour and became drowsy.

On neurological examination left homonymous hemianopia, left upper motor neuron facial weakness and left hemiparesis were found. The hemiparesis progressed to hemiplegia during the first hour of observation and then slowly improved.

The CT scan disclosed an ill-defined low density area in the right frontal lobe. The lateral horizontal portion of the right middle cerebral artery (MCA) was demonstrated as a high density structure on the non-contrast scan (fig. 2A). Its density did not change after contrast injection but there was enhancement of the more medial aspects as well as other arteries (fig. 2B). The finding was considered to be intra-arterial clot within the lumen beyond the origin of the middle cerebral artery. Angiography revealed occlusion of the right internal carotid artery with a stump suggesting dissection (fig. 2C) and occlusion of the middle cerebral artery trunk beyond the first temporal branch (fig. 2D). The suprasylvian branches filled through leptomeningeal collaterals.

The patient was treated with heparin. The hemianopia and paresis of the left leg completely resolved
within 48 hours. A slow but significant improvement was observed in the left upper extremity function.

Case 3

A 43 year old man had an episode of left-sided weakness following three transient episodes of ipsilateral weakness in the previous ten days. Angiography at that time revealed a significant right middle cerebral artery stenosis. He was treated with aspirin.

Three years later he presented with signs and symptoms that led to the angiographically-proven diagnosis of left posterior cerebral artery occlusion. The stenosis of the right middle cerebral artery had progressed substantially in comparison to the studies done three years earlier. Because he had not had symptoms referable to that lesion for three years, he was excluded from randomization in the Cooperative EC/IC Bypass Study. Despite the lack of symptoms referable to the right middle cerebral artery stenosis an EC/IC bypass was carried out in view of the progression of the stenosis. The patency of the anastomosis was proven angiographically.

Fifteen months after surgery he experienced the acute onset of weakness in the left extremities which gradually improved. On examination slight weakness of the left hand and decreased cortical sensation in the left arm were found.

On CT scan a small area of low density involving the right internal capsule was seen. The horizontal segment of the middle cerebral artery was visualized on the non-contrast scan as a high density structure (fig. 3A). The angiogram demonstrated complete occlusion of the right middle cerebral artery. Prominent collateral circulation had developed from the anterior and posterior cerebral arteries through leptomeningeal anastomoses and through the dilated EC/IC Bypass (fig. 3B). The latter filled the lateral lenticulostralite arteries as well (fig. 3C). Over the ensuing weeks further improvement was observed in the function of the left arm, coincident with coumadin therapy.

Case 4

This 50 year old woman without identifiable risk factors for vascular disease and with a 20 year history of periodic headache noticed a sudden onset of flashing lights and blurring of the vision two days prior to the admission. The next day she awoke unable to move her left side.

On admission she was found to be slightly drowsy. She had left homonymous hemianopia, and dense left hemiplegia, including the face. Pinprick and vibration sense were diminished on the left arm.

CT scan revealed a large low density area in the right frontal region. The right middle cerebral artery was visualized as a high density structure on the non-contrast scan. Cardiac studies disclosed no obvious source for emboli. The patient refused angiography. Slow recovery of the leg function occurred in follow-up.
Case 5

This 73 year old man had a femoral embolus and been submitted to femoral-popliteal bypass surgery in the past. The day before admission he experienced blurring of vision in the right eye, and on the day of admission he developed paralysis of the left extremities.

On examination a left homonymous hemianopia and left hemiplegia including facial weakness were present. A large low density area involving the entire territory of the right middle cerebral artery was visualized in the CT scan. The horizontal part of this artery could be seen as a high density structure on the non-contrast scan, and two days later, he was returned to his local hospital for further care. The patient’s neurological status was considered too severe for further angiographic investigation.

Case 6

An 82 year old woman, in apparently good health, became unable to speak and developed a right-sided weakness. The symptoms were followed by clonic activity in the right extremities for one hour without loss of consciousness. On examination, global aphasia, right homonymous hemianopia, and paralysis of the right face, arm and leg were found.

Case 7

A 75 year old man with a history of hypertension and chronic atrial fibrillation secondary to ischemic heart disease was found collapsed in his car. On arrival in the Emergency Department, he did not respond to verbal stimuli and exhibited Cheyne-Stokes respiration. Blood pressure was elevated at 210/120 and bradycardia was noted. His eyes were deviated to the right, with neglect of the left visual field, and a severe paresis of the left extremities and face were present. The ECG showed evidence of old anteroseptal myocardial infarction as well as atrial fibrillation.

CT scan revealed a large low density area in the distribution of the right middle cerebral artery with mass effect. The horizontal portion of the right middle cerebral artery was visualized as a high density area on the non-contrast scan.

After 48 hours the patient’s neurological deficit

The CT scan revealed a large low density area involving the left fronto-temporal parietal and occipital lobes. The left middle cerebral artery was demonstrated as a high density structure on the non-contrast scan (fig. 4).

The patient’s neurological condition was considered too severe for angiographic investigation.
worsened, he became unresponsive and expired six days after the onset of symptoms. No autopsy was performed.

Case 8

A 60 year old man with known atrial fibrillation had an episode of vertigo and sweating followed by temporary loss of consciousness. On examination following arrival at the hospital he was seen to have a right IIIrd nerve palsy, left hemiparesis, left-sided loss of pain sensation, and left upper quadrant field defect.

CT scan showed a low density area in the right hemisphere involving the posterior cerebral territory. The right posterior cerebral artery trunk was visualized as a high density area on the non-contrast scan (fig. 5). Follow-up scan showed enhancement in the right occipital lobe, medial temporal lobe, and thalamus corresponding to the distribution of the right posterior cerebral artery. Patient improved motor function but remained with a partial IIIrd nerve palsy, decreased deep pain sensation, and the upper quadrant field defect.

Discussion

High resolution CT scanning is capable of demonstrating structures of the size of intracranial arteries. The Circle of Willis and adjacent branches may be well visualized in the contrast enhanced scan. However, because of variations in the arterial course and differences in the slice level, the non-visualization of an artery cannot be interpreted as a sign of occlusion. On
the other hand, the demonstration of a normal-sized vessel as a diffuse high density structure on the non-contrast scan can hardly be explained by any cause other than partial or total thrombotic vascular occlusion, thrombotic or embolic. Our three cases with angiographic studies support this premise. The Circle of Willis along with a thrombosed vein of Galen were visualized in a case of "brain death" reported by Eick et al. They attributed the visibility of the arterial structures to the general lucency of the brain. We believe that the arteries were well seen due to their thrombosis as well.

Blood pool density as commonly seen in arteriovenous malformations is slightly greater than normal brain density. The theoretical possibility exists therefore that a normal artery may stand out as a higher density if surrounded by pathological lucency. Arteries at the base of the brain are surrounded by low density CSF, but even in patients with cerebral atrophy the arteries do not exhibit the high densities visualized in the 8 patients described in this report. The cases reported here show a true increased density of the affected arteries, in comparison in each case to other visualized arteries of the same scans. In view of the difference between the high density affected arteries and lower density unaffected arteries in the appropriate clinical setting of acute stroke, the finding of increased density is most consistent with intraluminal clotted blood. The suggestion of the diagnosis of acute occlusion of major intracranial vessels on CT scan may be very useful for the early initiation of confirmatory investigations, such as angiography, which may then lead to the early initiation of therapy.

A negative finding does not exclude an occlusion. Aside from the exact level of the slices, the age of the thrombus is probably the most important factor in visualization of the vascular occlusion because of the well documented density changes observed in intraluminal clot.2

Editor’s Note: In accordance with Stroke policy, this article was guest edited by JP Mohr, University of Southern Alabama.

References
Ataxic Hemiparesis in Patients with Primary Pontine Hemorrhage

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SUMMARY Two patients with small primary pontine hemorrhage developed a syndrome identical to ataxic hemiparesis, one of the lacunar syndromes of Fisher. The possible mechanisms of the homolateral cerebellar signs, pyramidal signs, and dysarthria are discussed. Lesions of the ipsilateral pontine nuclei may be responsible for the homolateral ataxia.

IN 1965, Fisher and Cole described a cerebrovascular syndrome, they called homolateral ataxia and crural paresis, characterized by cerebellar-like ataxia, weakness and pyramidal signs involving the limbs of the same side, the leg more than the arm. In 1978, based on the clinicopathological results in three patients with this syndrome, Fisher identified an old infarct cavity in the basis pontis at the level of the junction of the upper one-third and lower two-thirds on the side opposite the neurological deficit as the causative lesion. He also proposed the designation "ataxic hemiparesis" for this syndrome. Although this syndrome has been recognized as one of the lacunar syndromes of Fisher et al., lesions other than lacunes, such as tumors or demyelinating processes, could conceivably produce the syndrome.

Recently we examined 2 patients with primary pontine hemorrhage, that had a syndrome compatible with the ataxic hemiparesis of Fisher.

Case Report

Case 1

A 55-year-old hypertensive man, noted the sudden onset of dysarthria and weakness of the left leg and the left arm following occipital heaviness and nausea on November 3, 1981. He was unable to walk because of unsteadiness. Three hours later he was admitted to Tokai University Hospital. On admission his blood pressure was 160/110 mm Hg. He was alert and well oriented. The neck was supple. The left eye was artificial due to trauma in his youth; visual fields and extraocular movement of his right eye were normal. No pupillary abnormalities were detected. There was fine horizontal nystagmus on left lateral gaze. Speech was dysarthric. The nasolabial fold on the left was flattened; mild left hemiparesis was present particularly in the distal portion of the left leg. Deep tendon reflexes were normal, but Babinski sign was positive on the left. Sensation was normal. There was left dysdiadochokinesis, and the finger-nose-finger test, shin-tapping test and heel-shin test showed impairment out of proportion to his weakness.

CT scan on the day of admission showed a small recent hematoma in the right dorsal part of the rostral basis pontis (fig. 1). ECG and EEG examination were normal. Retrograde vertebral angiography was normal.

After admission, nystagmus rapidly disappeared, and the dysarthria and the left hemiparesis decreased in severity. CT scan on November 26 was now normal. Though the ataxia on the left side gradually improved, slight cerebellar signs still persisted with positive Babinski sign at discharge (December 7, 1981).

Case 2

This 39-year-old woman had been well until the evening of March 20, 1982, when she noticed dysarthria following a floating sensation for several minutes. She could not pick up a coin using her left hand because of clumsiness. Shortly afterwards, she noticed unsteadiness in walking. On admission, about four hours after onset, her blood pressure was 180/110 mm Hg. She was alert and well oriented. Neck was supple. Oculomotor functions and pupils were normal. The left nasolabial fold was shallower and the tongue devi-
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