Inadvertent Intra-Arterial Contrast Agent Injection Mimicking Bilateral Occlusion of the Internal Carotid Arteries in a Patient With Suspected Stroke on Maximum-Slope, Nondeconvolution Perfusion Computed Tomography

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Background and Purpose—Inadvertent contrast agent injection in the left cubital artery may lead to decisively altered perfusion parameters in stroke CT. These effects have not previously been described.

Summary of Case—A 77-year-old woman with a suspected stroke underwent subsequent stroke CT imaging. No signs of intracranial hemorrhage or acute cerebral ischemia were noted on nonenhanced CT. Qualitative analysis of perfusion CT using the maximum slope model demonstrated an extremely delayed and decreased perfusion of the territories of the anterior circulation system and higher values of cerebral blood flow and blood volume of the posterior circulation system mimicking a bilateral occlusion of the internal carotid arteries. CT angiography revealed no relevant stenoses or occlusions of the internal carotid arteries. Intensive investigation into the potential causes of these controversial findings showed that the contrast medium was administered into the left brachial artery due to inadvertent arterial placement of the antecubital catheter.

Conclusions—It is important to be familiar with this unusual constellation of perfusion parameters to avoid diagnostic uncertainty in patients with an inadvertent intra-arterial application of contrast agent. (Stroke. 2009;40:e46-e49.)

Key Words: acute stroke ■ carotid stenosis ■ CT ■ radiology
showed higher values of cerebral blood flow and cerebral blood volume as compared with the anterior circulation with a trend of higher perfusion values on the left side (anterior versus posterior circulation: cerebral blood flow: 12.1 versus 293.7 mL/min/100 mL; cerebral blood volume: 0.8 versus 15.3 mL/100 mL). CTA from the vertex to the aortic arch was performed using 120 kV, 190 mAs, bolus-tracking at the descending aorta, 64×0.6 mm collimation, and maximum intensity projections. On the basis of a weight-adapted contrast agent protocol, 97 mL contrast agent was injected over the same cubital catheter at 4.8 mL/s followed by a saline bolus of 100 mL at 4.8 mL/s. CTA images demonstrated no significant stenosis or occlusion of the craniocervical arterial vasculature (Figure 2). The only remarkable pathology was an incidental 3-mm aneurysm of the anterior communicating artery.

**Diagnosis**
The strikingly reduced perfusion values in the anterior circulation with comparatively higher perfusion values in the posterior circulation were due to inadvertent arterial placement of the antecubital injection catheter into the left brachial artery.

**Discussion**
The seemingly controversial findings of PCT and CTA with PCT suggesting a bilateral occlusion of the internal carotid arteries and CTA demonstrating no significant stenoses or occlusions of the supraaortic arteries initiated an intensive investigation into the potential causes. In agreement with CTA, duplex ultrasound of the carotid and vertebral arteries performed at the intensive care unit demonstrated no significant stenosis of the supra-aortic arteries. We therefore assumed that the discrepant CTA and PCT findings had to be due to erroneous analysis of the PCT data. The review of the raw data of the dynamic PCT clearly demonstrated a prominent, isolated contrast enhancement in the vasculature of the posterior circulation in the early phase (first wash-in) followed by washout and minor, delayed enhancement in the
arteries of the anterior and posterior circulation after recirculation in a later phase (second wash-in; Figure 3A–B). A time density curve of the contrast enhancement measured in the reference vessel (superior sagittal sinus) showed an enhancement peak after 11 seconds (not including the initial scan delay of 4 seconds; Figure 4A). Normalized arterial input function demonstrated a first lower peak at 3 seconds (700 Hounsfield units) and a second, higher peak at 7 seconds (1100 Hounsfield units; Figure 4A). Later on, calculated time density curve of the posterior and anterior fossa also showed the additional, prominent first wash-in phase at the posterior fossa followed by a minor peak in both posterior and anterior fossa after recirculation (Figure 4B).

When comparing the calculated values with normal brain perfusion parameters, we noted a strong reduction of the time to peak of the arterial input function and an uncommon biphasic split of the peak.\(^1\)\(^-\)\(^4\) Reduction of the time to peak of the arterial input function can either be due to an extremely fast blood circulation time or to an arterial instead of a venous contrast injection. Blood gas analysis extracted from the antecubital catheter confirmed our suspicion of accidental arterial placement of the antecubital catheter. The contrast medium was therefore administered into the left brachial artery instead of into a cubital vein. The flow rate of 7 mL/s of the contrast injection caused retrograde flow into the left vertebral artery.

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**Figure 2.** CT angiography: homogenous contrast enhancement at the circle of Willis; no sign for occlusion or hemodynamically relevant stenosis. Incidental aneurysm.

**Figure 3.** Perfusion CT raw data: (A) isolated contrast enhancement in the posterior circulation system in the early phase (first wash-in). B. Delayed enhancement in the arteries of the anterior circulation in a later phase (second wash-in).

**Figure 4.** A, Time density curve (TDC, white line) measured in the superior sagittal sinus shows enhancement peak after 11 seconds. Automatically calculated arterial input function (AIF, gray line) demonstrates a first, lower peak at 3 seconds and a second, higher peak at 7 seconds. B, TDC of the posterior (1) and anterior (2) fossa (including parenchyma and vessels) show the prominent first wash-in phase in the posterior fossa at 10 seconds and a minor second wash-in phase after recirculation with a peak at 28 seconds (dashed vertical line) in the posterior and anterior fossa.
The fact that the arterial injection had no obvious influence on the CTA images could be explained by the combination of 2 factors. First, the significantly lower flow rate of the injection was apparently too low to achieve retrograde filling of the subclavian artery. Second, the automated, threshold-based bolus tracking at the descending aorta compensated the prolonged recirculation time of the contrast medium due to the peripheral, arterial injection. A 3-way valve connected to the intra-arterial catheter seems to be the reason why the arterial blood backflow was not noted when connecting the tube of the contrast injector. Electroencephalography revealed periodic lateralized epileptiform discharges. After anticonvulsive therapy was started and sedation was stopped, the patient recovered to her previous neurological status. A generalized seizure was considered to be the most likely explanation for the patient’s loss of consciousness. One day after inadvertent intra-arterial contrast medium application, there was no evidence of digital damage or ischemia in the affected arm.

It is important to be familiar with this unusual constellation of perfusion parameters to avoid diagnostic uncertainty in patients with an inadvertent intra-arterial application of contrast agent.

Disclosures
None.

References
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