ORIGINAL ARTICLE

Postictal deficit mimicking stroke: Role of perfusion CT

Déficit post-ictal simulant un accident vasculaire cérébral : place du scanner de perfusion

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Summary

Objectives. — To demonstrate the use of perfusion CT in patients presenting with a suspected diagnosis of stroke to avoid the administration of inappropriate thrombolytic therapy in stroke-mimicking conditions such as status epilepticus.

Material and methods. — We reviewed the imaging studies of four patients presenting with symptoms suggestive of stroke, but finally diagnosed with status epilepticus. Imaging was by a 16-section multidetector CT scanner using a protocol consisting of non-contrast CT, CT angiography and perfusion CT. Color-coded maps allowed calculation of the CBV (cerebral blood volume), CBF (cerebral blood flow) and MTT (mean transit time).

Results. — In all four cases, perfusion CT revealed increases in CBF and CBV as well as a decreased MTT, consistent with hyperperfusion linked to status epilepticus with focal deficit— in contrast to the hypoperfusion observed in stroke patients.

Conclusion. — The use of perfusion CT accurately detected hyperperfusion in status epilepticus presenting as stroke. In such cases, perfusion CT imaging avoided the administration of potentially harmful thrombolytic therapy to patients experiencing seizures due to different underlying etiologies.

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KEYWORDS
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Introduction

Stroke diagnosis can be clinically difficult [1,2]. Conditions that mimic stroke can account for up to one-fifth of patients [1] and, in acute settings, only imaging can help to make the correct diagnosis. In emergency cases, reperfusion therapy relies on non-enhanced CT (NECT) [1] to exclude hemorrhage, an absolute contraindication for thrombolysis. Thrombolytic therapy has been associated with hemorrhagic complications that can be potentially harmful in patients with stroke-mimicking conditions [3]. Our aim was to establish the importance of using perfusion CT (P-CT) in the radiological evaluation of stroke to avoid misdiagnosing and mistreating non-stroke patients with thrombolytic therapy, especially patients with epilepsy.

Material and methods

Imaging protocol

At our hospital, all patients with a suspected diagnosis of stroke seen within 3 hours of symptom onset undergo, without delay, a NECT scan followed by P-CT and CT angiography (CTA), using a multidetector row CT scanner (Philips MX 8000 16-section multidetector CT scanner). NECT of the brain is performed using sequential 3-mm-thick sections. P-CT images are acquired during the infusion of 40 mL of a contrast agent (Accupaque 300) at a rate of 5 mL/sec, covering the region of the basal ganglia with a z-axis coverage of 24 mm. CTA is performed from the aortic arch to the vertex with a slice thickness of 1.5 mm after infusion of 100 mL of contrast agent at 4 mL/sec. Using the commercial software MxView V5.0 (Philips Medical Systems), color-coded maps allow the cerebral blood volume (CBV), cerebral blood flow (CBF) and mean transit time (MTT) to be calculated.

Imaging analysis

The NECT images were examined for early signs of infarction or hemorrhage. Perfusion CT images were post-processed, and the CBF, CBV and MTT calculated and compared with those of the contralateral hemisphere. Finally, CTA images were analyzed using maximum-intensity projection (MIP) reconstruction and multiplanar reconstruction (MPR) techniques. In this study, the radiologists were not blinded to the patient’s diagnosis when reviewing the images.

Electroencephalography (EEG)

EEG was performed using surface electrodes within 24 hours of the patient’s admission to hospital. A senior neurologist analyzed the recordings.

Results

Imaging studies of four patients presenting with symptoms suggestive of hemispheric stroke were reviewed retrospectively to determine whether or not P-CT could prevent thrombolytic therapy being given to patients with stroke-mimicking conditions. All of the imaging studies were performed within 3 hours of symptom onset, and all patients were eligible to receive thrombolytic therapy. In these four cases, non-contrast CT performed within 3 hours of symptom onset revealed no major early signs of stroke (low signal density in more than 33% of the MCA territory), although perfusion anomalies were present in the same territory as the suspected stroke. CBF and CBV values were higher than in the contralateral hemisphere. The final diagnosis in all four patients was epileptic seizure of different causes.

Patient 1 was a 38-year-old woman with a known history of epilepsy who presented with sudden sensory and motor hemisindrome (left-sided) and headache. A clinical diagnosis of stroke was made. CT imaging revealed no early signs of infarction, and P-CT showed hyperperfusion in the right temporal lobe, with increases in CBF of 50% (64 mL/100 g/min) and in CBV of 40% (3.7 mL/100 g/min), and a 30% decrease in MTT (3.4 sec) in the pathological hemisphere, matching the location of the pathological changes seen on EEG 2 hours later. CTA was normal.

Patient 2 was a 77-year-old woman with a history of chronic alcoholism who also presented with a left-sided sensory and motor hemisindrome. Stroke was suspected, but there was no sign of an infarct on NECT. P-CT showed hyperperfusion of the right hemisphere, with increases in CBF of 50% (59 mL/100 g/min) and in CBV of 40% (5.5 mL/100 g/min) in the pathological hemisphere; the MTT was also 30% shorter (5.3 sec). Hypervascularization was seen on the CTA reconstructions (Fig. 1) of the right hemisphere. Follow-up MRI revealed right hippocampal sclerosis, and subacute encephalopathy with seizures in chronic alcoholism (SESA) syndrome was finally diagnosed. EEG waveform abnormalities were present in the right temporal area as revealed by the perfusion-imaging findings.
Figure 1 In this patient (# 2) with SESA syndrome, all of the perfusion maps show abnormal values in the right temporal lobe. Increases in CBF (a) and CBV (b) as well as the decreased MTT (c) reflect ictal hyperperfusion. Coronal MRI FLAIR images performed one week after the CT reveal the abnormal high signal intensity within the right hippocampus (d, arrow).

Patient 3 was a 79-year-old woman with a history of resection of a left frontal meningioma, two years earlier. She presented with an episode of right-sided hemiplegia and aphasia. Suspecting stroke, CT was performed, and NECT revealed a postoperative sequela of frontal meningioma resection. CTA showed marked hypervascularization of the left temporal lobe (Fig. 2) as did P-CT, with increases in CBF of 50% (68 mL/100 g/min) and in CBV of 35% (5.7 mL/100 g/min), and a decrease in MTT of 50% (2.5 sec) (Fig. 2). Her final diagnosis was simple partial seizures with postictal Todd’s paralysis. Six months later, the perfusion maps were the same during a recurrence of the Todd’s paralysis, probably induced by modifications of her antiepileptic treatment.

Patient 4 was a 75-year-old man with a history of multiple sclerosis who presented with left-sided hemiplegia after a short episode of loss of consciousness. Although he progressively recovered from the hemiplegia, stroke was suspected and the patient was referred for imaging before initiating thrombolytic therapy. CT images showed no hemorrhage, and P-CT revealed increases in CBV of 30% (6.9 mL/100 g/min) and in CBF of 50% (112 mL/100 g/min) in the right temporal lobe that matched the EEG findings. MTT values were decreased by 50% (3.6 sec) in the pathological hemisphere. CTA findings were not relevant.

Discussion

It is estimated that between 5 and 30% of cases identified as “brain attacks” are, in fact, due to stroke-mimicking conditions [3]. During the European Cooperative Acute Stroke Study II (ECASS II), 17% of all patients who received thrombolytic therapy were ultimately proven not to have suffered strokes [4]. Stroke mimics include diagnoses such as complex migraine, conversion disorder, epilepsy and Todd’s paralysis [3,5]. If stroke is suspected, NECT is not sensitive enough to accurately detect early parenchymal changes [1,6]. Moreover, the use of NECT alone can result in reperfusion therapy being given to patients with stroke-mimicking conditions. In contrast, MRI offers good accuracy and sensitivity in such cases [7]. However, MRI is not always available in emergency settings, despite its better results in determining final stroke size, localization of ischemic lesions, and distinguishing between stroke and stroke mimics.

The accuracy of P-CT in detecting acute stroke has been studied on numerous occasions [6] and is widely used for acute stroke management. Recent studies have investigated the use of P-CT not only when stroke is suspected, but also for seizures [8,9]. In our four cases, we observed cortical hyperperfusion as reflected by an approximately
50% increase in CBF and a 30–40% increase in CBV values, with a decrease of around 40% in MTT (Fig. 1). On the other hand, in cases of stroke, P-CT reveals hyperperfusion (reduced CBF and CBV, and prolonged MTT). It is generally accepted that an increase in CBF and CBV values is found in the seizure-onset zone as well as in the cortical regions affected by the spread of ictal discharges during the course of a seizure [8]. The focal cortical hyperperfusion found in all four patients may be attributed to the peri-ictal phase of status epilepticus. Similar findings have already been described using perfusion MRI [10–12] and P-CT [8]. In those series, all perfusion parameters were affected by the ictal status, but mostly the CBF and least of all the CBV. MTT was decreased by 30–50%, whereas a prolonged MTT is a sensitive indicator of acute ischemia secondary to large-vessel occlusion [9]. In two of our cases (Patients 2 and 3), hyperperfusion on P-CT was associated with an increased vascular pattern on CTA—MIP reconstructions corresponding to the seizure focus (Fig. 2). Such patterns have already been described with angiography [13,14] and MRA [10]. However, P-CT has limitations, among which the z-axis coverage is probably the most important, as P-CT only covers around 20–40 mm of the brain. Also, there are other causes that can produce similar patterns, including reperfused stroke and migraine, where P-CT shows hyperperfusion. In such difficult cases, CTA may be of help in determining whether or not an intracranial occlusion or vascular spasm is present. Although seizures at stroke onset are not common (7%), it is possible that a patient has had both a seizure and a stroke. CTA may again be a useful modality for differentiating Todd’s paralysis from seizure and ischemia by detecting intracranial occlusion (Patient 3). This may contribute to the decision to administer thrombolysis [15–18]. Thus, P-CT hyperperfusion does not exclude stroke with seizures.

Conclusion

A review of our four cases demonstrates the use of P-CT in stroke-mimicking conditions. In such cases, P-CT accurately detected hyperperfusion changes that were not compatible with stroke. NECT alone is insufficient prior to thrombolytic administration as changes in neurological status can be linked to stroke mimics. Larger prospective studies as well as the further technical development of multi-slice scanners with larger brain coverage are needed to assess the clinical utility of this modality in the diagnosis of stroke-mimicking conditions.

References