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Regional Cerebral Hyperperfusion Associated with Postictal Paresis

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Abstract

The pathophysiology of postictal paresis and other symptoms of Todd's phenomenon are not fully understood. Various hypotheses about its mechanism include exhaustive neuronal firing, desensitization, and active suppression. This case series demonstrates CT perfusion findings of two patients with postictal paresis that show hyperperfusion, increased cerebral blood flow, increased cerebral blood volume, and decreased mean transit time in the intracranial distribution corresponding with neuronal symptoms. These findings may suggest an active process requiring adequate perfusion during Todd's phenomenon.

Abbreviations:

(CTP)	CT perfusion
(CTA)	CT angiography
(CBF, CBV)	cerebral blood flow and volume
(MTT)	mean transit time

Introduction

Todd's paralysis is a postictal phenomenon first described in the nineteenth century,[1-2] defined as diminished motor ability lasting hours to days following a focal-onset seizure.[3] Other neurological deficits such as aphasia, neglect, and psychosis have since been attributed to Todd's phenomenon following partial and generalized seizures. Despite evidence of structural localization of the deficits,[4] the pathophysiology is unknown. Hypotheses include "neurotransmitter depletion, neuronal desensitization, altered local cerebral blood flow (CBF), and various forms of active inhibition."[5] These cases consistently demonstrate regional cerebral hyperperfusion associated with Todd's phenomenon by serial CT perfusion (CTP) studies. Hyperperfusion has been found in active seizure activity[6] but has not yet been described in relation to Todd's paralysis.

Case One

A 57-year-old man presented with aphasia and right hemiparesis of unknown symptom onset. A nurse at his group home discovered him unresponsive and unable to move his right side, a change from his vocal and ambulatory baseline. He had a history of traumatic brain injury and posttraumatic seizure disorder with occasional episodes of Todd's paralysis for the past 25 years. Upon arrival, he was alert but unable to consistently follow commands, repeat words, name objects, or answer questions. He opened his eyes spontaneously but didn't respond to visual threat on the right. Other cranial nerves appeared intact. He had minimal antigravity strength in the right upper extremity and no antigravity strength in the right lower extremity. He withdrew to painful stimuli on the left and grimaced on the right.

A stroke code was called, and the patient underwent protocol CT scanning, CT angiography (CTA), and CTP. Noncontrast head CT demonstrated no intracranial hemorrhage. The CTA of the head and neck was performed and 3D reconstructions and multiplanar image reformations were created using the Vitrea workstation. CTA showed no significant stenosis involving the major cervical or intracranial arteries.

The CTP study was administered according to protocol. Two rapid boluses of approximately 35--40 cc of nonionic iodinated contrast medium were used to perform dynamic intracranial CTP at two selected levels: one at the level of the lentiform nuclei and one at the level of the supraventricular white matter. Images were reconstructed at four selected levels and were reviewed on the Vitrea workstation. The CTP showed hyperperfusion of the left cerebral hemisphere, with increased regional CBF, increased cerebral blood volume (CBV), and decreased mean transit time (MTT) (see Figure).

The patient first recovered motor strength of right upper and lower extremities but remained uncooperative and aphasic. The pattern of recovery was typical according to his sister. Two days later, the patient was at baseline

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status and discharged to his nursing home with increased doses of anticonvulsants.

Approximately one year afterward, the patient presented again with expressive aphasia and right hemiparesis, with unknown symptom onset and last normal activity reported 4--6 hrs before presentation. Upon arrival, he was alert, unintelligible, and did not follow commands consistently. He had a leftward gaze preference and minimal movement of the right side. The patient underwent protocol imaging as above. Once again, CTA of the head and neck showed no stenosis of major arteries. CTP demonstrated increased perfusion with decreased MTT, and increased CBF and CBV in the left middle cerebral artery distribution.

Within hours, all extremities had returned to normal baseline strength. In stereotypic fashion, motor symptoms resolved within the first hospital day and aphasia eventually recovered in the days following admission. Anticonvulsant therapy was optimized, and the patient was discharged after four days.

Case Two

A 45-year-old man presented with altered mental status, aphasia, gaze deviation, and left arm weakness after being found on the street. A bus driver discovered him unresponsive and unable to move his left arm. He had a history of epilepsy, hypertension, and bipolar disorder. Upon arrival, he was confused and unable to consistently follow commands, repeat words, name objects, or answer questions. He had a left gaze preference. Other cranial nerves tested appeared intact. He had minimal antigravity strength in the left upper extremity. He withdrew to painful stimuli bilaterally.

The patient underwent stroke protocol imaging as above. Noncontrast CT showed no evidence of intracranial hemorrhage, mass effect, or hydrocephalus. CTA of the head and neck showed no stenosis of major cervical or intracranial arteries. CTP demonstrated hyperperfusion with decreased MTT, increased CBF and CBV.

Within minutes, all the patient's symptoms had returned to normal baseline. A 20-channel EEG was performed within an hour of arrival, which revealed a normal study with the patient awake and in stage-2 sleep. The patient was loaded with Dilantin, admitted for 24-hr observation, and discharged under stable condition.

Discussion

CTP findings in these patients were distinct from acute ischemic stroke and probably related to Todd's phenomenon. In all presentations, CTP showed increased CBF, increased CBV, and decreased MTT. These findings are similar to those of active seizure. However, since the patients were alert and able to follow some commands, postictal phenomenon versus ongoing ictal state was ruled in clinically. In the second patient, an EEG also ruled out nonconvulsive status epilepticus. These events revealed decreased MTT, implying that the ability to perfuse the brain is intact, which may be the key difference between Todd's paralysis and stroke. The consistent finding of hyperperfusion that is similar to active seizure may reveal our results as early postictal changes after residual ictal activity. These results may point to the active suppression theory of Todd's phenomenon.

With aphasia and right-sided hemiplegia, acute stroke is in the differential diagnosis and must be assessed rapidly as thrombolytic therapy is time sensitive. The use of CTA and CTP studies in stroke protocol has been established. A thorough review by König summarizes the status of brain CTP in acute stroke: CTP determines the extent of hypoperfusion involved in ischemic stroke with sensitivity of approximately 90% and no false positives. Although there is no numeric guideline dictating the classification of stroke by CBF and CBV, these values are clearly diminished in ischemic infarcts and are sufficient for diagnosis. CBF, which is inversely related to MTT, is decreased in both infarcted tissue and ischemic penumbra while CBV is decreased only in irreversibly damaged tissue. Thus, in acute left-sided stroke causing aphasia and right hemiplegia, hypoperfusion would be found and MTT would be augmented in the CTP with high likelihood of finding the offending vessel on CTA. The exact opposite was found in these patients' studies: hyperperfusion with increased flow and volume was found and MTT was diminished. None of the CTA studies showed occlusion anywhere in the head and neck vasculature.

After acute ischemic stroke was ruled out based on the CTP and CTA findings, the radiology report suggested seizure as a possible etiology. A case report by Royter et al. describes the importance of differentiating between ischemic stroke and nonconvulsive status epilepticus and postictal Todd paralysis, as clinical presentations may be similar or may precede the other. They describe a patient presenting with left hemiparesis and slurred speech who was found to have a short MTT, increased CBF and CBV on CTP. Bedside EEG confirmed ongoing abnormal neural firing consistent with status epilepticus, which was treated with phenytoin load.[6] This report appears to be the first to describe CTP findings in acute seizure, and the resulting values have since been

replicated.[8] However, consistent data regarding postictal states are still lacking. One case report showed that in one patient with aphasia and right hemiparesis secondary to Todd's paralysis, there was a "dramatic" decrease in CBF and CBV with no change in MTT.[5] These results are more similar to ischemic stroke than to seizure activity. Unlike our CTP findings, these results suggest a mechanism of neuronal excitotoxicity and exhaustion as the cause of Todd's paralysis.

The imaging studies performed in this case may have captured the pathophysiology of Todd's paralysis at different points of progression. The studies were not performed under ideal circumstances: an EEG was obtained in only the latter patient to unequivocally rule out status epilepticus (although clinical examination clearly suggested Todd's paralysis in the first) and baseline CTP studies were not obtained. Nevertheless, the consistency of these CTP findings associated with clinical Todd's phenomenon suggests that the findings of our series are accurate, and it is our hope that this case series can inspire and aid further investigations of a poorly understood phenomenon.

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