

Very Early Demonstration of Secondary Pyramidal Tract Degeneration by Computed Tomography

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Background While magnetic resonance imaging has revealed progressive changes in the pyramidal tract in accordance with histopathologic stages of wallerian degeneration secondary to a supratentorial lesion, computed tomography (CT) has only demonstrated a shrinkage of the pyramidal tract in the midbrain or pons during the chronic stage. We present a patient with frontoparietal subcortical hemorrhage in whom serial CT scans clearly demonstrated wallerian degeneration along the axis of the pyramidal tract early in the acute stage.

Case Description A 63-year-old man with a history of hypertension suddenly developed a deterioration of consciousness, transcortical mixed aphasia, and dense hemiplegia on the right side. CT scans revealed a massive intracerebral hematoma in the frontoparietal subcortices of the left hemisphere. Although initial CT did not detect any hypodense

areas along the left pyramidal tract below the hematoma, ill-defined areas of decreased density appeared in the posterior limb of the internal capsule, cerebral peduncle of the midbrain, and pontine base of the left side on day 13 after the stroke. These areas became well demarcated on day 22 and persisted thereafter.

Conclusions An extensive hematoma can interrupt the pyramidal tract fibers that arise not only from the motor cortex and caudal premotor cortex but also from the somatosensory and parietal cortices, allowing very early CT demonstration of wallerian degeneration of the pyramidal tract. (*Stroke*. 1994; 25:2287-2289.)

Key Words • cerebral hemorrhage • computed tomography • wallerian degeneration

Wallerian degeneration refers to the anterograde disintegration that affects an axon and its myelin sheath after injury to the proximal portion of axon or its cell body.^{1,2} Although Waller³ described this degenerative process observed in the hypoglossal and glossopharyngeal nerves, the same term has been applied to similar nerve fiber changes occurring in the central nervous system. Abnormalities in involved axons precede changes in the myelin sheath. Myelin sheaths collapse, and the lamellae become loose.¹ Daniel and Strich⁴ studied wallerian degeneration in the spinal cord in baboons and found that myelin sheaths break up into ellipsoids within 48 hours but retain the staining properties of normal myelin for many weeks. McCaman and Robins⁵ reported that breakdown of myelin into simpler lipids was not observed until 100 days after section of rabbit optic nerves. The time course of wallerian degeneration varies greatly. It occurs more rapidly in peripheral nerves than in the central nervous system, in larger caliber fibers than in smaller ones, in unmyelinated fibers than in myelinated fibers, in younger animals than in older ones, and in mammals than in cold-blooded animals.^{1,6}

The recent advent of neuroimaging in clinical practice has provided demonstrable proof of wallerian degener-

ation in brain-damaged humans. Magnetic resonance imaging (MRI) has demonstrated pyramidal tract changes in patients with supratentorial lesion, in accordance with the histopathologic stages of wallerian degeneration mentioned above, by using T₁-weighted, T₂-weighted, and/or proton density-weighted images.^{2,7-12} In contrast, most studies involving computed tomography (CT), the most prevalent diagnostic technique to date, have only revealed shrinkage of the pyramidal tract in the midbrain or pons in the chronic stage.^{13,14}

We recently treated a patient with massive subcortical hemorrhage in whom serial CT scans demonstrated marked wallerian degeneration of the pyramidal tract early in the acute stage.

Case Report

This 63-year-old, right-handed Japanese man had a history of hypertension and diabetes mellitus. He suddenly developed a deterioration of consciousness, language difficulties, and motor weakness of the right extremities just after attending a meeting on December 1, 1993. He was hospitalized for initial care and was diagnosed as having intracerebral subcortical hemorrhage. He was transferred to our institute on December 4, 1993.

On admission the patient was mildly drowsy. Neurological examination disclosed a transcortical mixed aphasia, neglect for the right hemispace, and right-sided dense hemiplegia involving the face. Muscle tonus was flaccid on the right. The deep tendon reflexes were exaggerated, and the plantar response was extensor on the right.

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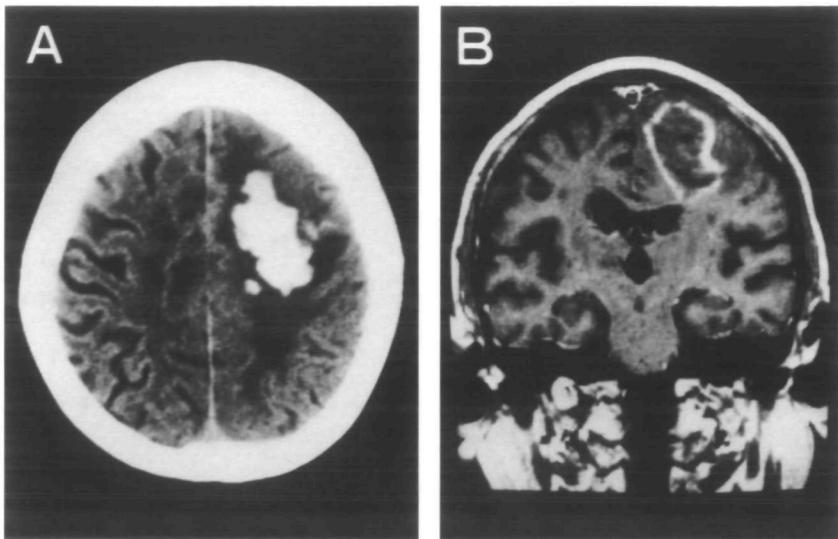


Fig 1. A, Axial computed tomographic scan on admission (fourth day after ictus) showing a massive intracerebral hematoma in the frontoparietal subcortices of the left hemisphere. Hypodense areas consistent with cerebral edema are observed extensively around the hematoma. B, Magnetic resonance images. Coronal T₁-weighted spin-echo images (repetition time, 600 milliseconds; echo time, 17 milliseconds) after intravenous injection of gadolinium-diethylenetriamine pentaacetic acid (0.1 mmol/kg of body weight) on the sixth day demonstrate the areas of very slightly decreased intensity with a halo of increased intensity. The mass is believed to interrupt the fibers converging to the corona radiata from the precentral gyrus and paracentral lobule of the left side.

CT scan using an X-force scanner (Toshiba) with a slice thickness of 10 mm on admission (fourth day after stroke onset) revealed a massive intracerebral hematoma in the frontoparietal subcortices of the left hemisphere (Fig 1A). Hypodense areas consistent with cerebral edema were observed extensively around the hematoma. Scattered old foci of decreased density were present in the bilateral basal ganglia or thalamus. No areas of abnormal density were found along the left pyramidal tract (Fig 2, top row). MRI studies were carried out on the sixth day after onset, using a 1.5-T system (Siemens Corporation). Axial T₁- and T₂-weighted and proton density-weighted images were obtained with spin-echo pulse sequences. T₂- and proton density-weighted images revealed the mass in the left frontoparietal subcortices to be of decreased intensity, and T₁-weighted images found the mass to be of

very slightly decreased intensity with a halo of increased intensity. Postcontrast coronal T₁-weighted images revealed a rim of increased intensity surrounding the mass, which appeared to interrupt the fibers converging to the corona radiata from the precentral gyrus and paracentral lobule of the left side (Fig 1B). T₂-weighted images revealed areas of markedly decreased intensity in the left thalamus and bilateral putamen, as well as scattered small areas of increased intensity in the basal ganglia and other subcortical regions. The former areas seemed consistent with old intracerebral hematomas and the latter ones with old foci of lacunar infarcts.

Sequential CT scans were performed on the 9th, 13th, and 22nd days. Ill-defined hypodense areas in the posterior limb of the internal capsule, cerebral peduncle of the midbrain, and basis pontis on the left emerged on the 13th day (Fig 2, second row). These areas of

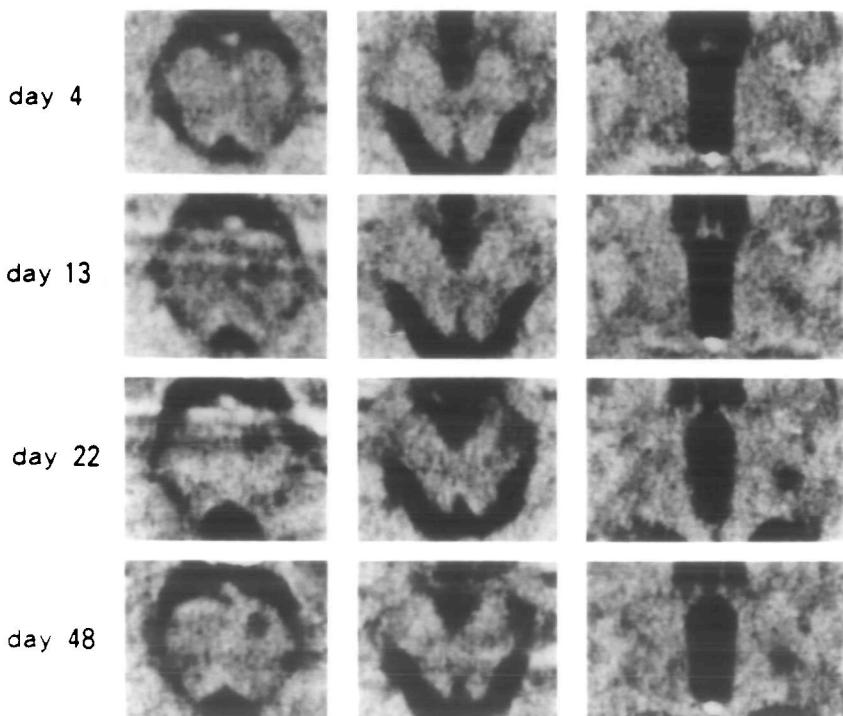


Fig 2. Sequential computed tomographic scans at the levels of the internal capsule (right column), cerebral peduncle (middle column), and pons (left column). No areas of abnormal density appear along the axis of the left pyramidal tract on the fourth day after the initial ictus. Ill-defined hypodense areas emerge in the posterior limb of the internal capsule, cerebral peduncle of the midbrain, and basis pontis on the left side on the 13th day. These areas of decreased density become well demarcated on the 22nd day and are still present on the 48th day.

decreased density along the left pyramidal tract became well defined on the 22nd day (Fig 2, third row).

The patient's language disturbance gradually improved, but the dense hemiplegia was unchanged. The patient suffered from a recurrent intracerebral hemorrhage in the right frontal opercular subcortex on January 2, 1994 (the 33rd day after the initial ictus). The hypodense areas along the axis of the left pyramidal tract remained, with gradual attenuations revealed on CT scans on the 33rd, 34th, 35th, 41st, and 48th days (Fig 2, bottom row). The patient was transferred to another hospital on January 18, 1994 (the 49th day).

Discussion

Sequential CT findings in our patient were quite characteristic. As shown in Fig 2, although initial CT did not reveal any hypodense areas along the left pyramidal tract below the level of hematoma, ill-defined areas of decreased density appeared in the posterior limb of the internal capsule, cerebral peduncle of the midbrain, and pontine base of the left side on the 13th day after stroke onset. These areas became well demarcated on the 22nd day and persisted thereafter during our observation period. These findings were consistent with secondary (wallerian) degeneration of the pyramidal tract.

Wallerian degeneration can be detected as shrinkage of the ipsilateral brain stem after damage to the supratentorial corticospinal tract in patients with chronic hemiplegia of duration of more than 1 year.^{13,14} Exceptionally, Kuhn and coauthors⁸ described a single case of decreased attenuation in the mesencephalon and pons on CT scans. Kameyama and associates¹⁵ reported a patient whose CT scans demonstrated focal low densities in the cerebral peduncle and pontine base 1 month after middle cerebral artery occlusion. To our knowledge, the present report describes the earliest demonstration of wallerian degeneration of the pyramidal tract by CT.

Why was such an early CT change apparent in our patient? Most of the fibers of the pyramidal tract originate in the motor cortex and caudal premotor cortex, but 20% stem from the somatosensory and parietal cortices.¹⁶ Pennock and colleagues¹² reported on two adults in whom areas of increased signal intensity in the internal capsule were detected on T₂-weighted MRI within 2 weeks of the onset of frontoparietal infarction, and they attributed the rapid onset of changes to the relatively sparse myelination of the parietopontine tract. Fumeya and Hideshima¹¹ reported that T₂-weighted MRI revealed early abnormalities of the corticospinal tract within 1 week of ictus in patients with spontaneous intracerebral hemorrhage. They attributed these early MRI findings to the occurrence of acute endoneuronal edema in the corticospinal tract fibers. Early CT changes in our patient might correspond to the above-mentioned MRI abnormalities, on the basis of the same underlying mechanism, although we could not obtain repeat MRI. From the present case, we conclude that a massive hematoma, spreading out through the frontoparietal subcortices, extensively in-

terrupts the fibers from the cerebral cortex to the spinal cord, thus causing the very early manifestation of wallerian degeneration of the pyramidal tract in the internal capsule, midbrain, and pons demonstrated by CT.

Prospective studies with CT and MRI should be conducted to detect wallerian degeneration early and to clarify its mechanism in acute stroke patients.

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