

Magnetic Resonance Imaging of Wallerian Degeneration in Stroke

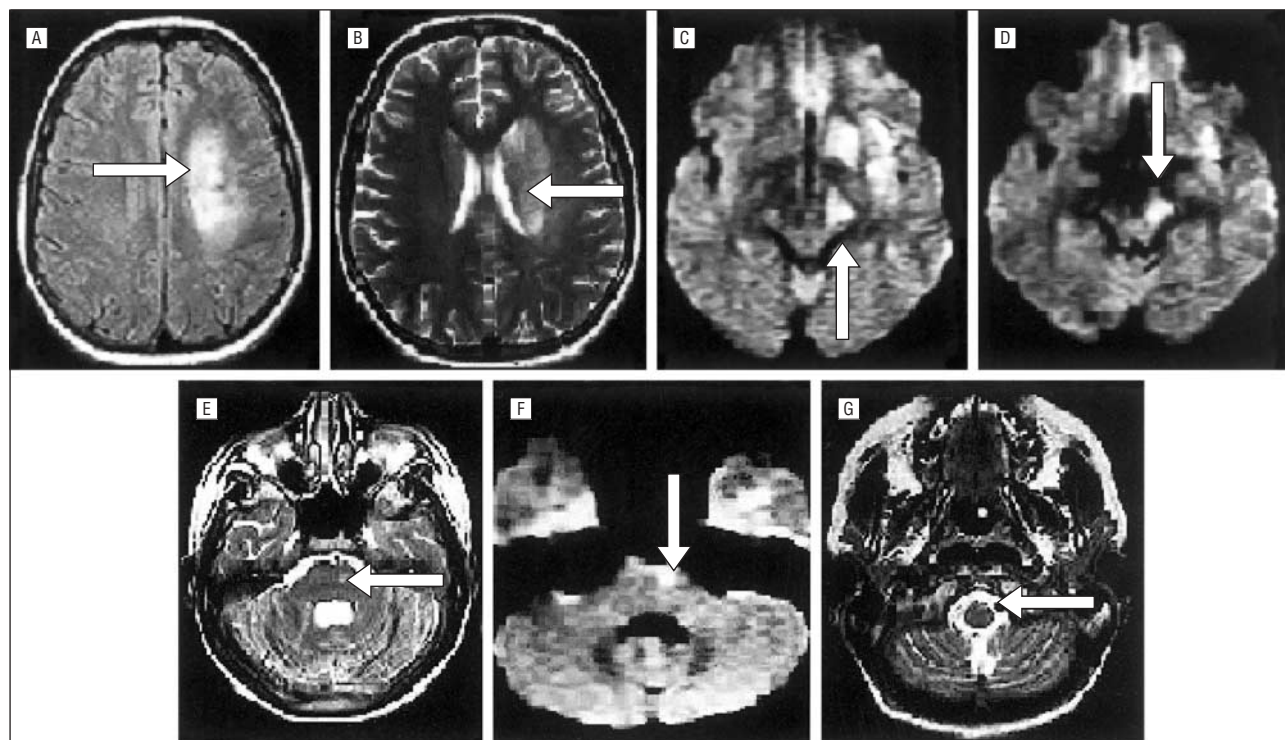
A 17-YEAR-OLD GIRL had sudden onset of right hemiparesis and dysarthria. The patient had no significant medical history. She had transcortical sensory aphasia and severe right hemiparesis. Tendon reflexes were more brisk on the right side than on the left; the neurologic examination was otherwise normal. Cranial computed tomography showed a left parietal and frontal subcortical infarction. After an extensive workup, the mechanism of the infarct remained undetermined. Cranial magnetic resonance imaging performed 9 days after the ictus disclosed a left ischemic stroke involving the frontal and parietal subcortical areas (**Figure**, A and B). Hyperintensity on

diffusion- and T2-weighted images (**Figure**, C-G) as well as hypointensity on T1-weighted images were present in the corticospinal tract, from the internal capsule to the medulla. Hyperintensity on fluid-attenuated inversion recovery (FLAIR) images was present from the internal capsule to the pons. Hypointensity was demonstrated in these areas on the apparent diffusion coefficient map, representing restriction to water diffusion.

COMMENT

Wallerian degeneration is the anterograde degeneration of axons and their myelin sheaths caused by proximal axonal or cell body lesions.¹ Myelin protein breakdown, increase in water content, and de-

crease in lipid-protein ratio as well as myelin lipid levels have been described in degenerating axons.² Wallerian degeneration may begin within 1 week of damage to a fiber tract, and demyelination can continue during the next 6 months. An increase in signal intensity on T2-weighted images and/or a decrease on T1-weighted images, attributed to wallerian degeneration, have been described as early as 8 days but most often at least 4 weeks after corticospinal injury.¹ A hyperintense signal in the corticospinal tract at the cerebral peduncle on diffusion-weighted imaging has been reported in one patient within the first 3 days after middle cerebral artery infarction.² In the present case, signal intensity abnormalities were ob-



FLAIR (fluid-attenuated inversion recovery) (A) and T2-weighted image (B) show hyperintense signal in middle cerebral artery infarction (arrows). Hyperintense signal is seen on T2- (E and G) and diffusion-weighted (C, D, and F) images in the region of the left corticospinal tract (arrows) at the level of the cerebral peduncle (C and D), pons (E and F), and medulla (G).

served on FLAIR and diffusion-, T1-, and T2-weighted images throughout the corticospinal tract, from the internal capsule to the medulla.

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