A 22-year-old female was seen in the emergency within one hour of acute onset of right sided headache followed by weakness of the left side of body. On neurological examination, she was mildly drowsy, had forced right gaze deviation, dysarthria, left hemiplegia and left hemisensory loss. Computed tomography (CT) scan revealed early ischemic changes in the right middle cerebral artery (MCA) territory. The CT angiography done showed evidence of dissection of the supraclinoid segment of the right internal carotid artery with reduced flow distally into the MCA, which was confirmed by a conventional angiogram. In view of the intracranial carotid dissection, the patient was not treated with intravenous tissue plasminogen activator. Magnetic resonance imaging (MRI) of the brain done on the next day revealed evidence of acute ischemic lesions in the right MCA and anterior cerebral artery territory on diffusion-weighted imaging (DWI), with normal brainstem. [Figure 1] A repeat MRI performed 13 days after ictus showed hyperintense signal on DWI in the right cerebral peduncle which was hypointense on apparent diffusion coefficient (ADC) map suggestive of Wallerian-like degeneration. [Figure 2] The signal changes were less conspicuous on T2-weighted images. She had antigravity strength in the left leg but remained weak in her left arm at one month.

Wallerian degeneration is defined as peripheral axonal degeneration after transection of a nerve trunk itself. In the central nervous system, Wallerian–like axonal degeneration occurs similarly; it is most frequently seen affecting the corticospinal tract after injury to the motor cortex.1 Wallerian-like degeneration can begin within one week of damage to the fiber tract and the demyelination can continue for the next six months. The availability of DWI and diffusion tensor-imaging (DTI) has facilitated early detection of Wallerian-like degeneration, within 2-14 days after ischemic stroke.2-4
Wallerian-like degeneration develops through four stages. The first stage is characterized by physical disintegration of the axons and myelin sheaths. This acute white matter tract injury is associated with cessation of energy dependant axo-plasmic transport and cytotoxic edema. The DWI identifies this earliest stage of Wallerian-like degeneration as a hyperintense signal which is hypointense on ADC map. But the conventional imaging will be normal during this stage. The second stage takes place 4-14 weeks after stroke and is characterized by rapid destruction of myelin and is seen as hypointense signal on T2 and proton density images. In the third stage gliosis occurs, which gives a hyperintense signal on T2 and fluid attenuated inversion recovery (FLAIR) sequences. The stage 4 or end stage occurs several years later and reveals volume loss from atrophy of the white matter tracts. Axonal degeneration of the middle cerebellar peduncle also has been reported following pontine infarction.5,6 Diffusion tensor-imaging in particular the parallel and perpendicular diffusivity has been shown to reflect axon and myelin pathologies respectively in the optic nerve of a mouse model of retinal ischemia.7 Studies in humans have shown that, compared to DWI, DTI may be a better tool to stage Wallerian-like degeneration, as it correlates better with axonal and myelin integrity.1-8 The exact prevalence of Wallerian-like degeneration after acute stroke is not known. A study by Castillo and Mukherji2 detected evidence of Wallerian-like degeneration in 2 of the 11 (18%) patients who underwent DWI within 72 hours of ischemic stroke of the pyramidal tract after ischemic stroke reflects severe pyramidal tract damage and may slow the functional recovery and has been associated with persisting impairment of motor functions, as was observed in our patient.9-11 Further prospective studies are needed to establish the correlation of the DWI changes of Wallerian-like degeneration and the functional outcome to determine its utility as a prognostic marker after stroke. Identification of these lesions is important, as these new lesions on follow-up imaging should not be misinterpreted as new ischemic lesions.

REFERENCES