SHORT REPORT

Wallerian degeneration of the inferior cerebellar peduncle depicted by diffusion weighted imaging

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Wallerian degeneration of the inferior cerebellar peduncle has never been demonstrated on imaging studies. We describe a case in which it was depicted by thin slice diffusion weighted imaging. Location to the inferior cerebellar peduncle was confirmed by a fibre tracking method.

Certain groups of neurones in the central nervous system may atrophy or degenerate after injury to their axons or cell bodies, or after the death of interconnected afferent or efferent cells. The former phenomenon is referred to as wallerian degeneration and the latter as transneuronal degeneration. Magnetic resonance imaging (MRI) has been used to study both conditions in various parts of the central nervous system, including the corticospinal tract,^{1–5} the optic radiation,^{6,7} the corpus callosum,^{8–9} the limbic circuit,^{10–11} the brain stem,¹² and the spinal cord.^{13–14} In this report, we describe a case of an infarct in the lateral medulla and concomitant abnormal hyperintensity along the inferior cerebellar peduncle. Location to the peduncle was confirmed using a fibre tracking method. To our knowledge, this is the first report to show wallerian degeneration of the inferior cerebellar peduncle using MRI.

Figure 1 (A) Axial T2 weighted image through the medulla shows only a subtle area of increased signal at the left lateral medulla (open arrow). Slices through the inferior cerebellar peduncle fail to show any significant abnormality. (B) Contiguous thin slice diffusion weighted imaging in 2 mm section thickness showing tiny hyperintense foci along the location of the left inferior cerebellar peduncle (solid arrows). The subtle area of increased signal at the left lateral medulla is also shown (open arrow), as in panel A. (C) Note that the contralesional inferior cerebellar peduncle also has subtle hyperintensity. Tiny hyperintense spots at the tegmentum pons represent the medial lemnisci. Fibre tracking images of this patient show that the above mentioned hyperintensity is present along the course of the inferior cerebellar peduncle.

CASE REPORT

The patient was a 50 year old man with nausea and vomiting of sudden onset, associated with forward leaning posture and inability to walk. He was referred to our hospital seven days after the onset of vomiting. On admission, his blood pressure was 154/66 mmHg and his pulse was 90 beats/min. He was alert and well oriented. Extraocular movements were normal; however, gaze evoked nystagmus was noted when he looked to the left. He had a positive Horner's sign on the left and ipsilateral facial sensation was diminished. The left soft palate was poorly elevated. Hypalgesia and hypothermoaesthesia were observed on the right side of the body below the neck, while proprioception and vibratory sensation were preserved. The finger-nose test was clumsy on the left side. On the basis of this typical constellation of signs, a diagnosis of left lateral medullary syndrome (the Wallenberg syndrome) was made.

Magnetic resonance imaging was done 12 days after the onset of symptoms. The images were obtained using a whole body 1.5 Tesla imager (Gyrosan Intera, Philips Medical Systems, Eindhoven, Netherlands). Diffusion weighted imaging was undertaken as previously described.¹⁵ In brief, a single shot EPI technique was used, with time of repetition (TR) = 6000, time of echo (TE) = 88, flip angle = 90°, and six motion probing gradient (MPG) orientations. A b value of 800 s/mm² was used, with six times image averaging. The recorded data points were in a 128 × 37 matrix using the parallel imaging technique. The reduction factor for the parallel imaging technique was 2, which results in true resolution equivalent to a matrix size of 128 × 74. The data were zero filled to a final resolution of 128 × 128. Thirty six slices were obtained with a thickness of 2 mm without interslice gaps.

Transaxial T2 weighted images showed only a subtle area of increased signal at the left lateral medulla, which was more apparent on diffusion weighted imaging (open arrows in fig 1, panels A and B). This imaging finding confirmed the diagnosis of lateral medullary syndrome. When scrutinising the images for an additional area of signal abnormality, we identified a small hyperintense focus at the lateral wall of the

Figure 2 Three dimensional depiction of fibre tracts showing the relations between the cerebellar peduncles. Inferior cerebellar peduncle (yellow), middle cerebellar peduncle (blue), superior cerebellar peduncle (bright orange), motor (violet), and sensory (green) tracts are depicted. They are arranged for stereo view. A colour coded vector map shows the direction of the local fibres, represented by red (left-right), green (anterior-posterior), and blue (superior-inferior).

fourth ventricle (solid arrows in panel B). As it appeared that this structure had longitudinal orientation in the craniocaudal direction, we hypothesised that the signal abnormality was occurring as a result of wallerian degeneration in the inferior cerebellar peduncle secondary to the lateral medullary infarct. Fibre tracking was then done using thin slice diffusion weighted imaging, which revealed a clear correspondence of the signal abnormality and the location of the inferior cerebellar peduncle (fig 1C). Three dimensional images of the superior, middle, and inferior cerebellar peduncles were generated to clarify the locations of these structures (fig 2).

DISCUSSION

Wallerian degeneration is usually not observed until four weeks after the onset of symptoms, when conventional MRI (generally T2 weighted imaging) is used.^{1,2} Earlier depiction of wallerian degeneration has recently been reported using diffusion weighted imaging.^{16,17} Kang *et al* reported two cases of wallerian degeneration of the corticospinal tract identified by diffusion weighted imaging¹⁶; both cases were scanned 12 days after symptom onset. Castillo *et al* examined 11 patients who were scanned within 72 hours after the onset of symptoms and found wallerian degeneration in two.¹⁷ Our patient was scanned 12 days after symptom onset, and this longer interval before diffusion weighted imaging was done may have facilitated the visualisation of wallerian degeneration. Nevertheless, to the best of our knowledge, wallerian degeneration of the inferior cerebellar peduncle has not been shown previously by MRI. This probably reflects the subtle nature of the finding. High resolution images would be essential in detecting such abnormalities; in the case described here, diffusion weighted imaging was undertaken using 2 mm slice thickness. Image averaging of six times yielded a sufficient signal to noise ratio, even with this thin slice section.

Using the fibre tracking method, we were able to depict the inferior cerebellar peduncle, which further confirmed the localisation of the abnormal signal in that structure. It is worthwhile pointing out that the fibre tracking was successful even in the presence of an infarct involving part of the inferior cerebellar peduncle. The fractional anisotropy of the infarcted tissue may, in some cases, be maintained or even increased at the acute/subacute stage of infarction,¹⁸ enabling fibre tracking to be done at the earliest stage of the infarct. In our patient, the fractional anisotropy at the infarcted area of the inferior cerebellar peduncle measured 0.68 ± 0.25 .

Neuroanatomically, the inferior cerebellar peduncle consists of fibres of the olivocerebellar and dorsal spinocerebellar tracts. The function of the olivocerebellar system is coordination of movement. The dorsal spinocerebellar tract carries axons from the dorsal column that contain rapidly conducted proprioceptive information. Fibres of the dorsal spinocerebellar tract ascend ipsilaterally, enter the cerebellum through the inferior cerebellar peduncle, and then spread through all areas of the cerebellum. On diffusion weighted imaging, the dorsal spinocerebellar tracts can be observed as a subtle area of hyperintensity. Such hyperintense fibre tracts can be seen in several other places, including the corticospinal tracts, the medial lemniscus, the superior cerebellar decussation, and the superior cerebellar peduncles. The exact cause of the hyperintensity is not fully understood, but it may be due to a high degree of anisotropy, as the source images of diffusion weighted imaging show hyperintensity of the tracts depending on the direction of motion sensitising gradients. Among the ascending pathways that pass through the medulla, the spinocerebellar tract is one of the fastest conducting, carrying

Ia and Ib fibres. These fibres are large and well myelinated. This histological characteristic of the spinocerebellar tract is conceivably the source of the strong anisotropy.

The clinical implications of wallerian degeneration of the corticospinal tract depicted by MRI have been established, and it is known that degenerated corticospinal tract cases are associated with a worse motor outcome.⁵ Whether wallerian degeneration of the inferior cerebellar peduncle has similar clinical implications is yet to be determined. Finally, the hyperintensity of the inferior cerebellar peduncle may mimic a new infarct focus, so care needs to be taken to interpret the findings correctly.

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