High signal intensity on T1 weighted MRI of the anterolateral column of the spinal cord in amyotrophic lateral sclerosis

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Abstract
Objective—To investigate MRI abnormalities in patients with amyotrophic lateral sclerosis

Methods—Fourteen patients with amyotrophic lateral sclerosis underwent MRI of the head and spinal cord using T1 and T2 weighted images. Forty age matched controls (29 with other neurological diseases, 11 with non-neurological diseases) underwent MRI of the cervical spinal cord using T1 and T2 weighted images.

Results—In all the control patients, the signal intensity of the posterior column was equal or slightly hypointense compared with the anterolateral column of the cervical spinal cord on T1 weighted images. However, eight of 14 patients with amyotrophic lateral sclerosis showed pronounced high signal intensity in the anterolateral column of the spinal cord on T1 weighted MRI, which also disclosed high signal intensity of the intracranial corticospinal tract in two of the 14 patients. T2 weighted MRI demonstrated high signal intensity of the lateral corticospinal tract of the spinal cord in two, high signal intensity of the intracranial corticospinal tract in five, and low signal intensity of the motor cortex in six of the 14 patients. Two of the 14 patients showed no abnormal findings on MRI.

Conclusions—High signal intensity of the anterolateral column of the spinal cord in patients with amyotrophic lateral sclerosis is a new imaging abnormality and may be useful for the diagnosis of this disease.

Keywords: amyotrophic lateral sclerosis; magnetic resonance imaging; cervical spinal cord

Amyotrophic lateral sclerosis is a progressive degenerative disorder of the upper and lower motor neurons and is of unknown aetiology. Magnetic resonance imaging has been reported to be useful for detecting lesions of the upper motor neurons in amyotrophic lateral sclerosis.

T2 weighted MRI may show areas of high signal intensity in the corticospinal tract extending from the corona radiata to the brainstem and cervical spinal cord in patients with amyotrophic lateral sclerosis, that are thought to reflect degeneration of the corticospinal tract.

Lesions in the motor cortex in amyotrophic lateral sclerosis are often seen as a low signal intensity of the motor cortex on T2 weighted MRI. However, this is not found in all patients with the disease. As we have incidentally noted signal abnormalities on T1 weighted MRI in patients with amyotrophic lateral sclerosis we compared both imaging methods.

Materials and methods

Patients
Fourteen patients with amyotrophic lateral sclerosis (10 men and four women; aged 41–78 years) were studied by MRI. The duration of illness ranged from four months to 48 months with a mean of 17 months. Initial symptoms were weakness and muscular atrophy in the arms in 10, in the legs in two, and bulbar palsy in two patients. The diagnosis of amyotrophic lateral sclerosis was established clinically from the patients’ history and results of physical and electrophysiological examinations. Other possible diagnoses were excluded by the use of appropriate laboratory tests that included examination of CSF and measurement of leucocyte lysosomal enzyme activity. All the patients showed active, chronic denervation potentials on EMG, with evidence of reinnervation in three or four limbs. Sensory and motor nerve conduction tests were normal except for reduced amplitudes of the compound action potentials.

The control group consisted of 40 age matched patients with progressive spinal muscular atrophy (four), cervical spondylotic myelopathy (10), Parkinson’s disease (five), spinocerebellar degeneration (five), HTLV-I associated myelopathy (three), subacute combined degeneration of the spinal cord (two), and non-neurological diseases (11).

The scans were visually analysed by three radiologists who were unaware of the patients’ clinical histories.

MRI
T1 and T2 weighted MRI of the brain and the
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Figure 1. (a) Axial T1 weighted images (TR: 500 ms, TE: 25 ms) of the cervical spinal cord at the C4–5 segment of a control (a 48 year old man). The signal intensity is almost equal in the anterolateral and posterior column of the cervical spinal cord. (b) T2 weighted image (TR: 3000 ms, TE: 100 ms) at the same level. The signal intensity of the grey matter is higher than white matter.

Figure 2. (a) Axial T1 weighted images (TR: 500 ms, TE: 25 ms) of the cervical spinal cord at the C4–5 segment, and (b) the lumbar spinal cord at the Th 12-L1 segment of a patient with amyotrophic lateral sclerosis (a 53 year old man, duration of illness four months). High signal intensity is present in the anterolateral column in both segments.

cervical spinal cord were obtained for all the patients with a 1.5 T superconducting MRI unit (Signa, General Electric, Milwaukee, WI, USA). Lumber MRI was also performed on six patients. T1 weighted images of the brain and spinal cord were obtained with a spin echo pulse sequence with a repetition time of 500 ms and an echo time of 25 ms.

T2 weighted images of the brain and spinal cord were obtained with a spin echo pulse sequence with a repetition time of 3000 ms and an echo time of 100 ms. The data acquisition matrix was $256 \times 192$, the field of view was 20 cm, and contiguous 5 mm axial slices were obtained.

Results
The posterior columns were isointense or slightly hypointense compared with the anterolateral columns of the cervical spinal cord on T1 weighted images in all the control patients (fig 1a). The white matter of the cervical spinal cord could be seen as a low signal intensity area compared with the grey matter on T2 weighted images in the controls (fig 1b), except for a patient with subacute combined degeneration of the spinal cord who showed high signal intensity in the posterior column of the cervical spinal cord on T2 weighted images.

Imaging disclosed mild to moderate compression of the cervical spinal cord in 10 patients with cervical spondylotic myelopathy; none of them showed high signal intensity at the sites of compression on T2 weighted images.

Eight of the 14 patients with amyotrophic lateral sclerosis showed very high signal intensity in the bilateral anterolateral column compared with the signal intensity in the posterior column of the cervical spinal cord on T1 weighted images (fig 2a). In the eight patients with amyotrophic lateral sclerosis, high signal intensity areas in the anterolateral column of the cervical spinal cord were more pronounced at the lower (C4, C5, C6) than at the higher (C1, C2, C3) cervical level, but were not seen above the medulla oblongata. Four of the eight patients with amyotrophic lateral sclerosis had high signal intensity areas in the lumbar spinal cord on T1 weighted images as well as in the cervical spinal cord (fig 2b).

Two of the eight patients with amyotrophic lateral sclerosis had high signal intensity in the dorsolateral area of the cervical spinal cord on
T2 weighted images, which corresponds to the lateral corticospinal tracts. Four patients with amyotrophic lateral sclerosis who had cervical spondylosis showed compressive lesions in the cervical spinal canal on T1 weighted images, but showed no signal abnormalities on T2 weighted images. Five of the 14 patients with amyotrophic lateral sclerosis (two patients with and three patients without a high signal intensity area in the cervical spinal cord) had high signal intensity areas in the corticospinal tract of the brain that extended from the corona radiata to the cerebral peduncle on T2 weighted images. Two of the five patients had high signal intensity areas in the posterior limb of the internal capsule and corona radiata on the axial T1 weighted images as well as the T2 weighted images, which correspond to the intracranial corticospinal tract.

Six of the 14 patients with amyotrophic lateral sclerosis had low signal intensity in the motor cortex on T2 weighted images (Fig. 3). Two of the 14 patients with amyotrophic lateral sclerosis had no abnormal findings on MRI.

The interval between the onset of symptoms and MRI was within 11 months in eight patients who showed high signal intensity in the anterolateral column on T1 weighted images. The eight patients were all young (41–54 years old) and had rapid courses. Electrophysiologically they showed active denervation potentials. Upper motor neuron signs were not seen in six of these eight patients.

Discussion
It is not difficult to diagnose amyotrophic lateral sclerosis given the characteristic combination of upper and lower motor neuron signs in the absence of sensory, sphincter, and eye movement abnormalities, but it may be difficult to diagnose when upper motor neuron signs, such as increased tendon reflexes and spasticity, are masked by the apparent lower motor neuron features. It may also be difficult to differentiate the disease from progressive spinal muscular atrophy and primary lateral sclerosis, especially in the initial stages.

Abnormal signal intensities on MRI of the corticospinal tract and low signal intensities of the motor cortex have been reported on T2 weighted images of some patients with amyotrophic lateral sclerosis. Although these MRI findings are not always seen, they are considered to reflect degeneration of the upper motor neurons and are useful in the diagnosis of amyotrophic lateral sclerosis.

We focused on T1 weighted MRI in the cervical spinal cord. High signal intensity was seen in the anterolateral column of the spinal cords of patients with amyotrophic lateral sclerosis, but was absent in patients with other diseases, including progressive spinal muscular atrophy.

High signal intensity in the anterolateral column of the spinal cord on T1 weighted images was more frequent in patients with amyotrophic lateral sclerosis (eight of 14 patients) than other MRI abnormalities such as low signal intensity of the motor cortex (six of 14 patients), high signal intensity of the intracranial corticospinal tract (five of 14 patients), high signal intensity of the spinal cord on T2 weighted images (two of 14 patients), and high signal intensity of the intracranial corticospinal tract on T1 weighted images (two of 14 patients). However, there was no apparent correlation between the severity of the corticospinal tract signs and the appearance of high signal intensity in the spinal cord on T1 weighted images of patients with amyotrophic lateral sclerosis. High signal intensity of the spinal cord on T1 weighted images tended to be found in the relatively early stage of the disease (the interval between the onset of symptoms and MRI was within 11 months for all the eight patients with abnormalities) and four had no other abnormalities on MRI.

Pathological studies in amyotrophic lateral sclerosis have shown that degeneration of the corticospinal tract is generally most pronounced at the cervical spinal cord level and is associated with abundant macrophages containing sudanophilic lipid in the anterolateral columns. Degeneration is seen in the corticospinal tract and also other descending tracts in the anterolateral column of the spinal cord, and there is axonal swelling due to the accumulation of intraaxonal neurofilaments present ultrastructurally in the corticospinal tract.

Some form of disorganisation of the white matter tracts or changes of the central grey matter of the spinal cord including the anterior horns under pathological circumstances in amyotrophic lateral sclerosis would increase the signal intensities of the anterolateral column, which includes lateral corticospinal tracts plus central grey matter, on T1 weighted images.

We speculate that the pathological changes seen in amyotrophic lateral sclerosis—such as lipid laden macrophages, and accumulation of intra-axonal neurofilaments—and toxic
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