The occurrence of ATLL in a patient with HAM is extremely uncommon and has been described in only three other patients.  

It has been shown that the viruses isolated from patients with HAM and ATLL are identical in their genomic composition. The HTLV-1 proviral carrier rate has been estimated to be 15% in the general population in Japan, and 5% in the Caribbean. The lifetime risk of developing ATLL if infected with HTLV-1 is 2% to 5% with an interval of about 30 years between acquiring the infection and developing symptoms. On the other hand the lifetime risk of developing HAM/TSP has been estimated to be 0-25%. Familial clustering is observed in individuals and families as well as in patients with ATLL. The degree of immune responsiveness is related to host genetic influences and the existence of HAM associated haplotypes and ATLL associated haplotypes has been suggested. Moreover, in vitro studies in patients with HAM have shown a high lymphocyte proliferation rate, spontaneously as well as in response to stimulation with both HTLV-1 and HTLV-1 specific antigens, compared with asymptomatic carriers or patients with ATLL. Also, the virus integration site into the host genome in HAM is random, whereas it integrates at a very specific locus in ATLL. The monoclonal integration of proviral DNA in ATLL consists of the long terminal repeat 5′3′ "tax" gene, the product of which induces interleukin 2 receptor expression and T cell proliferation.

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MATTERS ARISING

Magnetic resonance spectroscopic study of parkinsonism related to boxing

We read with interest the paper by Davie et al reporting a study of proton magnetic resonance spectroscopy (MRS) in the striatum of boxers with parkinsonism. They reported a significant reduction in the absolute concentration of N-acetyl-aspartate (NAA) in the putamen and globus pallidus in the boxers with a parkinsonism syndrome compared with patients with idiopathic Parkinson's disease and controls. They speculate that the reduced NAA may result from neuronal loss in the corpus striatum secondary to head trauma. In support of this hypothesis reference is made to their previous study in which it was reported that NAA is reduced in the lentiform nucleus in patients with striatogniral and olivopontocerebellar variants of multiple system atrophy compared with patients with idiopathic Parkinson's disease and controls.

This interpretation may be too simplistic. We have recently performed a study using MRS in 10 patients with idiopathic Parkinson's disease with motor response fluctuations on chronic levodopa treatment (satisfying the United Kingdom Brain Bank clinical criteria for Parkinson's disease) and seven healthy age matched controls using a voxel size of 4 ml centred on the putamen and one cerebellar hemisphere. We found a consistent and significant decrease of NAA/creatine and NAA/choline ratios in the putamen in patients with idiopathic Parkinson's disease but not in controls. The choline/creatine ratio between controls and idiopathic Parkinson's disease patients and cerebellum were unchanged suggesting that the changes seen were due to changes in MR-visible NAA itself. Repeat studies in two patients three months later, with regions of interest centred on the putamen bilaterally, showed similar reductions in the observed NAA signal.

These findings contrast with the results reported by Davie et al and raise several questions about the importance of localised changes in brain NAA in idiopathic Parkinson's disease and related disorders. Firstly, the exact positioning of the region of interest and voxel size are both likely to be crucial. The spectra analysed by Davie et al were obtained from a voxel centred on the globus pallidus and striatum, whereas our was restricted to the putamen. Striatal pallidal degeneration is a feature of multiple system atrophy, but not (so far as is known) of idiopathic Parkinson's disease. The findings of Davie et al thus may reflect the pathological changes in the pallidum rather than in the putamen. Certainly, it is not possible to conclude from the study of Davie et al that striatal NAA concentration is unchanged in idiopathic Parkinson's disease compared with multiple system atrophy and other related disorders.

Similarly, in the study of Hoshouser et al in which there were no significant differences in "striatal" NAA/choline ratios in patients with idiopathic Parkinson's disease between 51 and 70 years of age with controls, the region of interest was centred wholly on the globus pallidus, not in the putamen and a much larger voxel size (8 ml) was used. Furthermore, Hoshouser et al reported that choline/creatine ratios in idiopathic Parkinson's disease and controls were in the normal range and it is surprising, therefore, to note that they found significant reduction in NAA/choline and not NAA/creatinine ratios in idiopathic Parkinson's disease. Thus at present conclusions on the relevance of changes in NAA concentration or NAA/creatinine ratios in the "striatum" in idiopathic Parkinson's disease, multiple system atrophy, and other neurodegenerative disorders such as progressive supranuclear palsy or parkinsonism in boxers are premature.

Our finding of reduced NAA/creatine and NAA/choline ratios in boxers with Parkinson's disease may reflect a functional change, loss of nigrostriatal dopamine terminals, or loss of intrinsic striatal neuropeptides, or a combination of these factors. Diagnostic error is another possibility as the presenting clinical picture of idiopathic Parkinson's disease has an accuracy of 82% but the reduction in NAA/creatine ratios were consistent in most of our patients diagnosed with the disease.

Further work is needed to establish the best paradigms for acquiring spectra in idiopathic Parkinson's disease and related disorders to decide whether striatal (putaminal) NAA really is reduced, and to understand the significance of reductions in NAA in terms of neuronal dysfunction and pathology.

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