off 1-49 MoM). When all measures were included in a discriminant function with stepwise selection of variables, hippocampal height, width of the choroid fissure, and width of the temporal horn alone allowed correct classification of 22 of 26 cases (85% sensitivity) and 20 of 21 controls (95% specificity).

Our data suggest that measures of medial temporal lobe atrophy focusing more precisely on the hippocampus might be useful aids in the diagnosis of Alzheimer’s disease even in the early stages of the disease.

Reversible cortical oedema mimicking cortical dysplasia in mitochondrial disorder

Partial seizures are invariably associated with focal brain pathology. Optimised MRI in the evaluation of these patients for surgery has greatly improved the detection of a spectrum of lesions. Imaging findings, however, are not always specific for a particular pathology, and may transiently mimic a fixed structural lesion.

We briefly report the clinical and laboratory findings of a patient who had occipital lobe epilepsy since the age of 18. Seizures consisted of frequent and prolonged visual auras (hallucinations, palinopsia, photopsia) followed by reversible but progressive visual loss, as well as complex partial and secondary generalised seizures. Clinical findings were that of slurring, severe hearing and visual loss, and dysarthria. The patient’s mother had had a stroke at the age of 34 followed by seizures and dementia.

Visual evoked potentials showed abnormal latencies. Monitoring with EEG showed non-specific interictal slowing of background rhythms and focal seizures arising from left and right occipital lobes.

A mitochondrial cytopathy was confirmed by the presence of ragged red fibres and abnormal mitochondrial ultrastructure in the muscle biopsy. Magnetic resonance imaging during a period of increased seizure activity showed thickening of the cortical ribbon of the right parieto-occipital cortex in T1 weighted images. Increased signal was seen in the T2 weighted sequences (fig 1). A diagnosis of cortical dysplasia was considered and the patient was referred to our centre for surgical evaluation. Repeat MRI five months later no longer showed the lesion (fig 2).

Retrospectively, it became apparent that the abnormality was due to transient cortical oedema associated with focal status epilepticus and not a fixed structural pathology of the cortex. Reversible cortical abnormalities have been shown by MRI in generalised and partial status epilepticus. The appearance may be misdiagnosed as a neoplasia or ischaemic stroke if the transient nature and temporal relation to status epilepticus is not recognised. The unusual linear and peri-cortical extent of the reversible signal abnormality in our patient led to the initial misdiagnosis of a migrational disorder and the patient was referred for evaluation for surgery for epilepsy. Further investigation showed a mitochondrial disorder in our patient and the transient cortical oedema may indeed be secondary to altered cerebral energetics and the pathogenetic mechanisms causing severe seizures in this condition. This case report illustrates that transient functional MRI abnormalities may mimic fixed structural lesions.

We gratefully thank Professor Einhaupl and Dr Lalor of Universitätliklinikum Charité, Berlin; Dr Findeis and Dr Hecker, Hoffnungstaler Anstalten, Lobetal; and Dr Zierke, Universitätlinikum Poliklinik für Epileptologie, Bonn for providing valuable diagnostic and imaging information.

Ingrid Tuxhorn

Hans Holthausen

Alois Ebner

Epilepsy Centre Bethel,

Department of Preclinical Diagnostics, Mara 1,

Marauen 21,

D-33516 Bielefeld,

Germany

Sohey Noachtar

Neurologische Klinik und Poliklinik,

Klinikum Großhadern,

Ludwig-Maximilians-Universität,

München, D-81778 München,

Germany

Absence of SCA1 mutation in idiopathic cerebellar ataxia

Idiopathic cerebellar ataxia refers to a group of sporadically occurring cerebellar degenerations of unknown aetiology, which are clinically characterised by progressive ataxia with an onset in adult life. Neuro-pathological and clinical studies suggest that there are at least two types of idiopathic cerebellar ataxia. One group of patients presents with additional non-cerebellar symptoms, such as parkinsonism, autonomic failure, and pyramidal symptoms (idiopathic cerebellar ataxia-P). The underlying pathology in many of these patients is olivopontocerebellar atrophy. Those patients with idiopathic cerebellar ataxia-P who develop severe autonomic failure are subsumed under the broader category of multiple system atrophy. The other group of patients is clinically characterised by a pure
Reversible cortical oedema mimicking cortical dysplasia in mitochondrial disorder.

I Tuxhorn, H Holthausen, A Ebner and S Noachtar

*J Neural Neurosurg Psychiatry* 1994 57: 1439
doi: 10.1136/jnnp.57.11.1439

Updated information and services can be found at:
http://jnnp.bmj.com/content/57/11/1439.1.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/