

### Isolated palsy of the fourth cranial nerve caused by an intracavernous aneurysm

Sir: We report a patient with an isolated palsy of the fourth cranial nerve caused by a small laterally pointing aneurysm arising from the posterior part of the intracavernous internal carotid artery. We have been unable to find any previous reports of an intracavernous aneurysm causing a fourth nerve palsy alone without the subsequent development of other neurological abnormalities. A 52 year old secretary had been in good health until she woke one morning with a right temporal headache. This gradually cleared but 2 days later, while reversing her car, she felt giddy and nauseated and noticed double vision. This was more marked on gaze downwards and to the left.

On examination she proved to have an isolated weakness of the right superior oblique muscle but no other neurological abnormalities. Non neurological examination was unremarkable. She had no signs of cardiac or vascular disease and her blood pressure was 170/85 mmHg. A CT scan of the head (with and without contrast enhancement) was normal. A right carotid angiogram revealed a small laterally pointing aneurysm arising from the posterior part of the intracavernous segment of the right internal carotid artery (fig).

She was treated with atenolol 50 mg twice daily. Double vision soon disappeared and 6 months later no neurological abnormalities could be found.

An isolated fourth nerve palsy is rare, and may be congenital or acquired. The com-

monest causes of an acquired palsy are trauma, vascular disease, and tumours, either extrinsic to the nerve or arising from within it (schwannomas). Other causes include surgery, collagen disease, and demyelination. In about one third of cases, no clear reason for the palsy can be found.<sup>1,2</sup> In most cases assigned to the "vascular" category, there is evidence of generalised vascular disease and the palsy is presumed to have been caused by involvement of the vasa nervorum.<sup>1</sup> Compression of the nerve by an extrinsic vascular lesion such as an aneurysm is much less common and is usually associated with involvement of adjacent cranial nerves.<sup>2</sup> In a series of 1000 cases of external ocular palsy from the Mayo Clinic there were only two patients with isolated fourth nerve palsy caused by aneurysmal compression. One was a man aged 60 years with a large basilar aneurysm, the other was a patient with an intracavernous aneurysm which initially produced a fourth nerve palsy alone but later proceeded to a total ophthalmoplegia.<sup>1</sup>

We have been unable to find any other reported case of an intracavernous aneurysm causing a fourth nerve palsy in isolation and our patient differed from the Mayo Clinic case in that no other cranial nerve palsies appeared subsequently.

Intracavernous aneurysms arise from the lateral aspect of the intracavernous internal carotid artery at the points of origin of the smaller arteries to the dura and hypophysis, most often at the origin of the artery of the inferior cavernous sinus.<sup>3</sup> An aneurysm at this site will project laterally between the third and fourth nerves above and the sixth nerve and the first division of the fifth nerve below. The sixth nerve is usually the first to be compressed; by the time that other cranial nerves are affected the sixth nerve is invariably involved.<sup>4,5</sup> Presumably in our patient the small size of the aneurysm and its rather unusual position far back in the cavernous sinus permitted involvement of the fourth nerve in isolation. Remission of symptoms, perhaps aided by hypotensive treatment is not uncommon with intracavernous aneurysms. Although some intracavernous aneurysms expand until they compress all the neural structures within the sinus, others stabilise in size at an early stage so that the displaced nerves can recover function.<sup>5</sup>

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### Reversible lithium neurotoxicity at normal serum level may refer to intracranial pathology

Sir: Lithium may have neurotoxic effects at normal and abnormal serum levels. The symptomatology consists of cognitive decline accompanied by cerebellar, brainstem and pyramidal signs.<sup>1,2</sup>

We have found that the occurrence of neurotoxic phenomena at normal serum levels may point to hitherto undetected neurological pathology.

A woman, aged 75 years, was admitted in a dysphoric manic state. She had a history of mixed bipolar disorder (DSM-111R), and was treated with clopenthixol 20 mg bd, and promethazin 25 mg tid and 50 mg at night. Extrapyramidal side-effects prompted cessation of neuroleptics. Lithium was prescribed and 2 weeks later, when the serum lithium level was 0.7 mmol/l, progressive restlessness, agitation, feelings of desperation, disorientation, loss of memory and decorum developed. Neurological examination showed symmetrical hyperreflexia. The electroencephalogram (EEG) revealed marked diffuse and rhythmic slowing of the background, especially in both fronto-temporal areas, but predominantly on the left side. Cerebral CT revealed an area of lower density deep in the white matter of the right parietal lobe and widening of cortical sulci.

The clinical picture and possible diagnosis of white matter infarction prompted cessation of lithium therapy. Thereafter, both the clinical picture and EEG normalised within 6 weeks.

A woman, aged 53 years, was admitted with a major depressive episode and mood-congruent psychotic features (DSM-111R). She had experienced two similar episodes, diagnosed as mixed bipolar disorder with



Fig Right carotid angiogram showing intracavernous aneurysm.

rapid cycling with a periodicity of cycles of 3 weeks. She did not improve on combined antidepressant/neuroleptic treatment, amitryptiline, 25 mg tid and 75 mg at night, and zuclopenthixol, 4 mg four times a day. This treatment resistance led to the addition of lithium.

During the third week of lithium therapy, at the low dose of 600 mg sdd and stable serum lithium level of 0.6 mmol/l, sleeplessness, concentration difficulties, dysarthric speech and gait ataxia developed. Cessation of lithium led to a rapid reversal of neurotoxic symptoms within 2 weeks. Neurological examination, EEG, ECHographic investigation and CSF examination were unremarkable afterwards. Cerebral CT, indicated by this neurotoxic episode and persistent depressive symptomatology with apathy, however, revealed a frontobasal bilobar tumour, homogeneously enhancing, and situated in the midline, which proved to be a frontal meningioma.

Lithium is increasingly used in psychiatry, often in elderly patients, and based on recent findings that it is able to augment the effect of anti-depressants.

We have shown that the occurrence of reversible neurotoxic phenomena at normal serum lithium levels, which we have seen in several patients in the past 2 years, may point to a possibly treatable intracranial pathology. In one of our patients lithium neurotoxicity pointed to cerebral infarction, in the other to frontal meningioma.

These findings are in accordance with those of Bekker (1987) concerning lithium-augmentation in geriatric patients; he found that, on introducing lithium, 42% of his cerebrovascular-compromised patients, who had transient ischaemic attacks, cerebrovascular accidents or multiple infarcts in their history, developed signs of neurotoxicity, compared with 14% of patients without this pathology.<sup>3</sup>

Why this symptomatology occurs in these patients is unclear. One theory is that there is an abnormal affinity for lithium in pathological brain tissue,<sup>4</sup> which is based on showing that rat neuroblastoma and glioma cells are able to contain high levels of lithium.<sup>5</sup> This theory is corroborated by one report of a lithium concentration nearly thrice the serum level in human glioblastoma tissue.<sup>6</sup>

We hypothesise, that these findings speak in favour of the existence of a diminished capacity for lithium removal in pathologically transformed brain tissue. Such a diminished capacity might lead to islands of high lithium concentration, influencing the surrounding (normal) brain and giving rise to the specific phenomenon of lithium

neurotoxicity at normal serum lithium levels.

Though risk factors concerning neurotoxicity of lithium may be known,<sup>2</sup> neurologists and psychiatrists should be aware of a "lithium neurotoxicity sign", which may be observed at normal serum levels of lithium and which may point to potentially treatable intracranial pathology.

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We thank Prof R J van den Bosch, PH D for his advice.

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#### Thirst and compulsive water drinking in medial basal limbic epilepsy: an electroclinical and neuropathological correlation

Sir: A sensation of thirst and compulsive water drinking are unusual ictal phenomena that have been reported to occur in associa-

tion with complex partial epilepsy.<sup>1</sup> Previous studies have indicated an association of the ictal behaviour and epileptiform activity in the temporal lobe.<sup>1</sup> The following report describes a patient with stereotypical, reproducible clinical events consisting of an intense desire for water. Ictal scalp recorded EEG studies demonstrated right anterior temporal electrographic seizure activity and histopathology of the right hippocampus revealed hippocampal sclerosis. This case provides confirmatory evidence that an alteration in water intake may occur during partial or localisation-related seizures associated with pathology in the mesiobasal region of the temporal lobe.

A 39-year-old right-handed woman was evaluated for consideration of surgical treatment of her epilepsy. The patient developed generalised tonic/clonic seizure activity at 14 years of age. Growth and development were normal and the aetiology of her seizure disorder could not be determined. When aged 21 years she began to experience complex partial seizures that remained refractory to antiepileptic drug therapy. All of the patient's seizures were stereotypic and began with the sensation of "lightheadedness" and palpitations, followed by an urge to drink water. She would run to a sink to consume fluids, often by placing her head directly under the faucet. She could consume 6-8 glasses of water during a seizure. Every seizure episode was associated with thirst, even if the patient had recently completed eating and drinking. Subsequent to a seizure the patient recalled her desire to drink. The patient always kept a large pitcher of water next to her bed. She did not drink in response to a dry mouth or disagreeable sensation in the mouth or throat. If water was not available she would consume cola or juice. On one occasion she could not locate a source of water and ran in "terror", injuring herself. The ingestion of fluids did not appear to terminate her seizure activity. The patient was forced to surrender her position as a store clerk because of her bizarre drinking behaviour during her seizures. Previous diagnostic considerations had included psychogenic polydipsia, panic attacks, diabetes mellitus and diabetes insipidus.

The patient was admitted for prolonged extracranial EEG studies with audiovisual monitoring. Neurological examination was normal. Multiple seizures were recorded. The episodes began with a motionless stare followed by lip smacking and head turning to the left. The patient then placed her hands on her chest. At this time she would intermittently follow simple commands but not speak. She then pointed to her mouth and motioned