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 commonest 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.[1,2] 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.[1] 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.[2] 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.[1]

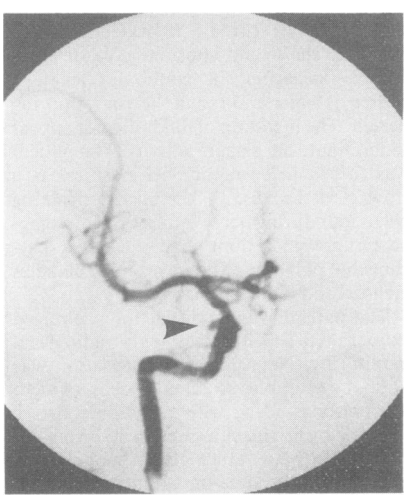

Fig *Right carotid angiogram showing intracavernous aneurysm.*

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.[3] 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.[4,5] 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.[5]

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References

1 Rush JA, Younge BR. Paralysis of Cranial Nerve iii, iv and vi. Cause and Prognosis in 1,000 cases. *Arch Ophthalmol* 1981;**99**: 76–79.
2 Rucker CW. The Causes of Paralysis of the Third, Fourth and Sixth Cranial Nerves. *Am J Ophthalmol* 1966;**61**:1293–8.
3 Barr HKW, Blackwood W, Meadows SP. Intracavernous carotid aneurysms: a clinicopathological report. *Brain* 1971;**94**:607–22.
4 Jefferson G. On the saccular aneurysms of the internal carotid artery in the cavernous sinus. *Br J Surg* 1938;**26**:267–302.
5 Meadows SP. Intracavernous aneurysms of the internal carotid artery. *Arch Ophthalmol* 1950;**62**:566–74.

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.[1,2]

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 diagnostis 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