abducens nerve palsy resolved gradually over 4 months.

The patient has one healthy son (age 8) and an unaffected mother, but his father and brother (the patient’s only sibling) had experienced cranial nerve palsies in the past. At the age of 39 (5/89), the brother developed a right abducens nerve palsy that resolved over 6 weeks. Three years previously he had had a myocardial infarction and subsequently required a 5-vascular coronary artery bypass. The patient’s father, an only child, experienced a left Bell’s palsy at age 49 and a left abducens nerve palsy at age 59 (7/84). At age 44 he was found to have 3 vessel disease by cardiac catheterisation. Both brother and father had normal neuroophthalmological examinations in January 1991.

A number of members had a thorough haematological evaluation for disorders predisposing to thrombosis including assays for cardiolipin antibodies, the Lupus anticoagulant (Russel viper venom time), prothrombin, partial thromboplastin, fibrinogen, fibrin split products, plasminogen, antithrombin III, protein C, and protein S. These tests were all normal or negative.

The familial occurrence of recurrent cranial nerve palsies remains uncertain despite extensive investigations including glucose tolerance tests, serologic testing, edrophonium (Tensilon) tests, cerebrospinal fluid analysis, single fibre electromyography, CT scan, and cerebroangiography.

Recently, haemato- and cardiovascular disorders predisposing to thrombosis have been associated with numerous neurological and systemic manifestations in young adults, including cerebral and myocardial infarction, deep venous thrombosis and migraine. 1, 2 Ophthalmic consequences (retinal arterial and venous occlusion, vitreous haemorrhage and ischaemic optic neuropathy) have also been reported in these patients. 3 Thrombosis in these disorders may be caused by a deficiency of one or more proteins integral to the regulation of clot formation (antithrombin III, protein C, protein S, fibrinogen) or by the presence of antiphospholipid antibodies (cardiolipin antibody, Lupus anticoagulant) that interfere with phospholipid dependent activation complexes (prothrombinase) essential to clot regulation. These tests were not available for the evaluation of previously reported patients with the syndrome of familial recurrent cranial nerve palsy. We believe that the pathophysiology of nerve damage in this syndrome as well as the vasculopathic because several of our patients had other evidence of vascular disease and vasculopathic disorders (diabetes, hypertension, coronary artery disease). These vasculopathic disorders are associated with cranial nerve palsies that present and improve in a similar manner. Our patients, however, had no laboratory evidence of antiphospholipid antibodies or deficiencies of coagulation cascade proteins and thus may have a separate disorder as well. Familial recurrent cranial nerve palsy may be caused by an as yet undefined vasculopathic process.

**Transient amnesia heralding brain stem infarction**

Transient global amnesia (TGA) is a syndrome of acute, transient memory disturbance with severe anterograde and retrograde amnesia but no neurological signs and preservation of personal identity. Vascular, epileptic, migrainous and hypoglycaemic causes have been implicated in its aetiology. 1, 2 A recent case control study has shown that most episodes of TGA are not associated with risk factors for ischaemic cerebral disease. Whilst ischaemic TGA may be familial, TGA is rarely associated with antiphospholipid antibodies (lupus anticoagulant) or other serological findings. Three months earlier, the patient had a complex partial seizure with impaired memory and a sense of unreality. This was followed by a 4-5 minute anterograde amnesia. The patient was fully orientated, with no focal neurological signs or a disturbance in consciousness, and there were no other abnormal neurological signs. An EEG performed at the height of his symptoms showed a slow wave transient in the fronto-temporal region. On the following morning (that is, 14 hours later) his ability to recall new information had returned to normal.

There remained, however, an amnesic gap for a short period of his train journey to the hospital on the previous day. Three days later, while still in hospital, he had a brief episode of dysphoria, numbness of the tongue, weakness and clumsiness of the left arm. There were no neurological abnormalities when he was examined shortly after this episode, which was diagnosed as a transient ischaemic attack. The next day he had a further transient episode of dysarthria and right hemiparesis, again with no signs on examination immediately after the event.

Twenty four hours later he became drowsy and developed periods of sleep which had evoked nyctagmus to the right with delayed adduction of the right eye on leftward gaze, preserved upgaze with upbeat nystagmus, consistent with a posterior fossa lesion. During this period he had transient weakness, diminished pharyngeal reflex, slow tongue movements and a dense right hemiparesis. CT scan showed slow attenuation in the right inferior cerebellar hemisphere and right occipital lobe consistent with infarction. MRI confirmed the diagnosis of brainstem stroke, with extensive high signal at the right cerebellar hemisphere, brain stem and medial part of right occipital lobe suggestive of occlusion of the right posterior inferior cerebellar artery and right posterior cerebral artery.

There was gradual neurological recovery over two months. He often appeared disorientated when discussed during this period but his mental state was otherwise considered to be normal and he performed satisfactorily on both verbal and visual recognition memory tests.

This patient presented with an episode of amnesia with features of TGA. 1 There was a witnessed attack of definite amnesia, resolving within 24 hours, without disturbance of consciousness, focal neurological signs, epileptic features or a recent head injury. During the attack the patient developed a clear anterograde amnesia with disturbance of long term memory without loss of personal identity, complex cognition or language. 3 He also exhibited the phenomenon of “meta-memory”, an awareness that memories ought to be readily recalled. Following recovery a small gap of retrograde amnesia remained. The only neurological sign noted was transiently blurred vision but examination was normal during the episode.

This case serves to emphasise that transient amnesia resembling TGA may occur as a manifestation of transient ischaemic attacks in the vertebrobasilar territory and that there is a risk of subsequent major ischaemic deficit.

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