stained with haematoxylin and eosin, periodic acid Schiff’s, Weil’s myelin, Klüver’s myelin, Nissl-creolk, Bodian, and Congo red. On microscopic examination loss of neurons was most conspicuously present in layer 5 of the left precentral gyrus, most prominent in the Betz cells. Neurrophagia (figure) and some proliferation of astrocytes was seen. The underlying white matter showed a decrease in the density of axons and slight gliosis. Other cortical areas, including the hippocampus amygdala, and Broca’s area showed no evident abnormalities. Although a single senile plaque was present, fibrillary tangles, Hirano bodies, Lewy bodies, amyloid deposition, and spongiform degeneration were not seen. The basal ganglia showed no abnormalities.

The clinical features of a right sided hemiparesis, slowly progressive in the course of three years, with only slight disturbances of intellect, in combination with postmortem evidence of non-specific loss of neurons in the motor cortex are indicative of local degenerative disease of the cerebral cortex. Alzheimer’s and Pick’s disease were ruled out on clinical and pathological grounds. In a series of six patients with progressive aphasia PET showed hypometabolism that was more extensive than the lesions visible on CT or MRI, as in our patient. The extensive involvement shown by SPECT was comparable with that in previously reported patients with isolated aphasia.

Our patient with an isolated hemiparesis of cortical origin, later accompanied by dysphasia, may represent a separate variety of a localised loss of cortical neurons, comparable with isolated motor aphasia. The mild disturbances of intellectual functions on neuropsychological examination are largely explained by the hemiparesis and dysphasia. Mesulam has also pointed out that such disturbances may result from the influence of the original lesion on the function of other regions of the brain.

Patients with localised cortical atrophy in non-dominant and without dementia have been described in several groups—namely, visuo-perceptual disorder; generalised apraxia; perceptuo-motor deficits, often combined with hemiparesis and behavioural changes; and progressive aphasia with unilateral extrapyramidal signs. The underlying pathological process in these localised cortical diseases is incompletely understood and not uniform. One patient with a slowly progressive aphasia showed (on biopsy) only non-specific and slight degenerative changes. One patient with a neuropsychiatric syndrome showed spongiform changes and mild gliosis in superficial layers of the prefrontal cortex. Three other patients with aphasia showed atrophy, spongiform changes, and astrocytosis, mainly in one or two layers of the cortex, without Pick or Lewy-type inclusion bodies. It seems that primary progressive hemiparesis is comparable with primary aphasia or extrapyramidal degeneration syndrome of the cerebral cortex. This variety of disorders follows a more protracted course than the more generalised dementias, and has no morphological characteristics to link it with Alzheimer’s or Pick’s disease.

We thank M H Dijkstra for referring the patient, Dr P J Koppelman for information about the patient and Dr P Maquet, for performing PET in 1988 in Liège, Belgium, L M P Ramos for his interpretation of the radiological series, and A F M M Verdonck for the neuropsychological examinations.

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Ventricular artery dissection mimicking migraine

We agree with Giroud et al that internal carotid artery dissection is a major cause of cerebral infarction in those under 50 years of age.1 We also suggest that spontaneous dissection of the vertebral artery is an important cause of ischaemic stroke in this age group, increasingly recognised with the advent of effective, non-invasive methods of diagnosis such as colour duplex ultrasound and MRI.2 We report a patient presenting with symptoms that would not necessarily have justified investigation by conventional angiography but who was found, by non-invasive means, to have had a vertebral artery dissection.

A 35 year old woman, previously well, suddenly experienced distorted vision while doing the housework. She described wavy, flickering lines in both eyes and hazy vision that persisted for about an hour. As the flickering faded she became aware that she could only see the right half of her husband’s face. This field defect persisted for about 30 minutes before her vision returned to normal. The next day she was aware of mild, diffuse headache and a stiff neck, which gradually improved over the next two weeks. There were no other associated symptoms and no history of trauma to the neck, although she had been playing in a competitive sports game earlier in the day. In the past, she had had hypertension while pregnant but there was no history of prior cerebrovascular events or of migraine. She did not smoke.

Examination two weeks after the onset of symptoms was normal other than slightly hyperextensible little fingers on both hands. Cardiovascular examination was normal; blood pressure was 130/80 mm Hg and no bruits were audible in the neck. Skin and other joints were normal and there were no focal neurological signs.

It was considered that her symptoms could have been caused by a vertebralbasilar transient ischaemic episode. Migraine was also a possibility. Blood tests, including blood count, glucose, cholesterol, lupus anticoagulant, antiphospholipid antibody concentrations, and syphilis serology were normal. Magnetic resonance imaging of the neck with axial, spin echo, T1 weighted images showed bright intramural thrombus with a very small residual lumen in the right vertebral artery (figure A). The abnormality was localised to a 2 cm portion of extracranial vertebral artery at the level of the C2 vertebra. Magnetic resonance angiography (MRA) showed disturbed flow within the vertebral artery at this level. The changes were diagnostic of vertebral artery dissection.

The patient was advised to continue aspirin for six months and has had no further symptoms. Repeat MRI, four months after the episode, showed no evidence of a persisting lesion (figure B).

Extracranial vertebral artery dissection, with occlusion or distal embolisation, is an important cause of transient ischaemic attack and stroke in patients under 50 years of age. Often there is no history to the neck or, when a history of trauma is present, it may be trivial. Epidemiological studies may have underestimated the true incidence of both carotid and vertebral artery dissection because of the requirement in the past for invasive angiography, with its attendant risk, to make the diagnosis. With the introduction of routinely available ultrasound and MR techniques, allowing non-invasive diagnosis, there is little doubt that more cases will come to light. It is interesting to note that the incidence of carotid dissection seems to be similar to that of aneurysmal subarachnoid haemorrhage.

This woman’s symptoms were “trivial” and the episode could easily have been diagnosed as an attack of atypical migraine with no further investigation performed.
Headache in lateral medullary infarction

Headache is a frequent, although under-emphasised, symptom of lateral medullary infarction and headache occurred in 22 of 41 patients with lateral medullary infarction. This frequency (54%) is much higher than that of cerebral infarction in general in large series (12 to 38%). We studied the incidence and features of headache in 34 patients with lateral medullary infarction.

These patients comprised 28 men and six women, mean age 54.4 (range 35 to 72) years. The diagnosis of lateral medullary infarction was confirmed by MRI for 30 of the patients. We interviewed all the patients regarding the presence, timing, localisation, side, and quality of their headaches. Angiography showed 4 of the 34 patients had angiography that included full neck views. Possibly some thromboses were secondary to dissection, especially in younger patients.

The head pain that accompanies lateral medullary infarction is likely to have two components; one engendered by the process occurring in the artery, the other the result of lesions in the central trigeminal system. In Fisher's series of 28 patients with head pain, 12 experienced only headache, six only facial pain, and 10 both. In our study, 26 of the 34 patients had headaches, 10 of whom also had facial pain. Facial pain in lateral medullary infarction usually occurs at the onset of stroke, has a characteristic nature (burning, stinging, unbearable, soreness), and is followed by numbness. Most of our patients who had both facial pain and headache could differentiate between them when carefully questioned. The high incidence of headache in lateral medullary infarction is not due to contamination by facial pain.

We conclude that headache is a very frequent accompanying or prodromal symptom in patients with lateral medullary infarction and that late life onset, occipital, throbbing headache heralds clinicoradiological evidence of lateral medullary infarction especially in the atherothrombotic subgroup.

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Supernumerary phantom limb after right hemispheric stroke

In a previous paper, we noted that there are few published cases of supernumerary phantoms after cerebral lesion. Yet whether this rarity reflects a genuinely low incidence of the phenomenon, a failure to inquire by the examiner, or an unwillingness on the part of patients to report a delusion that they themselves may regard as bizarre and threatening is unclear. We discussed the question of whether there is a coherent pattern in patients' descriptions of their experience (and a consistent neurological basis for the condition). We recently saw a second case of supernumerary phantom limb in whom the symptomatology was strikingly similar to our earlier case. The current patient was an 80-year-old woman who had suffered a stroke that resulted in thrombus formation in the vertebral artery. The patient's report included two phantom limbs in the right upper and lower extremities.
Vertebral artery dissection mimicking migraine.

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*J Neural Neurosurg Psychiatry* 1995 59: 340-341
doi: 10.1136/jnnp.59.3.340

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