Plain MRI of the neck and head, together with ultrasound, are probably the investigations of choice; MRA may prove helpful.

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Headache in lateral medullary infarction

Headache is a frequent, although underemphasised, symptom of lateral medullary infarction. Fisher reported headache in 22 of 41 patients with lateral medullary infarction.[1] This frequency (54%) is much higher than that of cerebral infarction in general in large series (12 to 38%).[2-5] 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 was performed on 14 of the patients.

Headache occurred in 26 patients (76%). It began in association with the neurological deficit in 13 patients, preceded the onset of stroke by one to five days in 10, and by 15 to 58 days in three. We limited further analysis to the 23 patients with headache in close temporal relation to the onset of the stroke. Nineteen of the 23 patients had lateralised headache on the side of the infarct, which was occipital or cervical in 15 (65%), frontotemporal in two (9%), diffuse in one (4%), and affecting the eye to forehead in one (4%). It was bioccipital in three patients (13%) and generalised in two (9%). Headache was throbbing in 13 patients (57%), eight of whose headaches were timed to their heart beat. It was described as dull or pressing in two (9%), intense in four (17%), and pricking in one (4%).

Proposed mechanisms of vascular occlusion were atherothrombosis in 22 patients, cardiogenic embolism in four, vasculitis of the vertebral artery associated with systemic lupus erythematosus in one, dissection of the vertebral artery in one, fibromuscular dysplasia of the vertebral artery in one, haemodynamic in one, and indeterminate in four. Headache was more common in association with atherothrombotic infarcts (16/22; 73%) than with cardioembolic infarcts (0/4; 0%). Of the 14 patients with angiographically confirmed lesions of the vertebral artery, including occlusion in eight patients (atherothrombotic six, dissection one, fibromuscular dysplasia one) and severe hypoplasia in six, 13 experienced headache. Nine angiograms included full neck views and five did not.

Headache was not related to the coexistence of cerebellar infarction or to its size. Of the 10 patients with cerebellar infarction in the territory of the posterior inferior cerebellar artery, six had headaches. Twenty of the 24 patients with infarction confined to the lateral medulla complained of headache.

Ten patients had pain in the eye, nose, and cheek, and all experienced headaches. All but one could distinguish facial pain from headache by its nature (burning, sore, unbearable), location, and appearance at the onset of stroke.

Our study confirms that headache is much more frequent in lateral medullary infarction than in cerebral infarction in general, although these studies suggest that headache is more likely to occur in posterior circulation ischaemia.[2-5] The underlying cause of headache in cerebral infarction is unknown. Various theories have been proposed[2-4]—namely, dilatation of an artery by a thrombus or embolus, dilatation of collateral circulation, release of a local endogenous chemical agent from the platelets, displacement of pain sensitive cerebral structures, and tension headache caused by emotional stress.

Our suggestion that headache in lateral medullary infarction is related to thrombus formation in the vertebral artery may support the platelet hypothesis. Headache in lateral medullary infarction was likely to occur in patients who had such conditions that resulted in thrombus formation especially in the vertebral artery (atherothrombosis, severe hypoplasia, and fibromuscular dysplasia), whereas none of the four patients with cardiogenic embolism complained of headache. In about half of the patients, headache preceded the onset of stroke and was throbbing in nature. In our study, dissection of the vertebral artery, which is a common cause of stroke in young people, might be underestimated because only nine 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 thought to have two components; one engendered by the process occurring in the artery, the other the result of lesion in the central trigeminal system.[1] 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.[1] 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 has clinical relevance as a warning sign of lateral medullary infarction especially in the atherothrombotic subgroup.

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Supernumerary phantom limb after right hemispheric stroke

In a previous paper,[1] 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 distressing is unclear.[2] The last issue raises the question of whether there is a coherent pattern in patients' descriptions of their experience (and a consistent neurological basis for the condition).

We have recently seen a second case of supernumerary phantom limb in whom the symptomatology was strikingly similar to our earlier case.[1] The current patient was an 80 year old, right handed man who was a retired teacher of mathematics. Against a background of hypertension of many years standing, he presented with a right hemispheric stroke on 3 March 1994. Examination showed a dense left hemiparesis, left sensory loss, left homonymous hemianopia, and gross left neglect. His Barthel score (an index of functional disability in daily living) was 4/20 (severely disabled). He ignored people on his left side, bumped into doors and furniture on the left and failed to make left turns when manoeuvring his wheelchair. Left neglect was also apparent when reading text; words on the left of the page were missed.

Computed tomography (6 April 1994) showed a very extensive area of low density in the distribution of the right middle cerebral artery. This is a mass effect with effacement of the right lateral ventricle and cortical sulci. There was no evidence of haemorrhage.

We examined the patient on 15 September 1994 (six months after his stroke) in the course of an investigation of anosognosia. The following extract is the pertinent part of the interview (P = patient; E = examiner):

P: I had a stroke.
E: How did the stroke affect you?
P: I don't drive and I don't do crosswords any more.
E: Anything else?
P: I carry a very heavy hand around with me.
E: How many hands do you have?
P: Would it surprise you if I said I had three?
E: Yes, it would. Do your hands work well?
P: My right hand is fine but the left arm is paralysed. The third one I carry it about and I sometimes place it here (*points with right hand to right upper leg*). It's concrete, but I can move the fingers.

E: Can you see it?
P: Yes, I can. It's very strange.
E: How many legs do you have?
P: Two.
E: Do they both work?
P: Yes, they do. It's the third hand that's peculiar. I don't want to talk about it.

As in the previous patient we have reported,[1] the supernumerary phantom occurs in the context of severe left hemiplegia and sensory loss associated with left neglect. In the earlier case, however, the lesion was a subcortical haemorrhage and there was no visual field deficit. Cognitively, both patients were fully aware that their left arm was paralysed and of the resulting handicap. Likewise, in both patients, there was a firm conviction of the "reality" of the third limb in conjunction with (rational) bafflement by the anomaly of the experience. Both men well realised that others would find their claim unbelievable, and were hence disinclined to discuss the issue at length. Why reduplication phenomena are typically (but not invariably) consequent on right hemispheric pathology[3] remains to be determined.

In reports of supernumerary phantoms prior to our own, the patients were usually seen in the acute phase, and it is generally assumed that, like anosognosia, the phenomenon remits rapidly. This patient, by contrast, shows that, like "ordinary" phantoms, the experience of a supernumerary limb can persist.

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Visually induced paroxysmal nausea and vomiting as presenting manifestations of multiple sclerosis

The protean manifestations and paroxysmal symptoms in multiple sclerosis are well described.[1] We report a patient with clinically definite multiple sclerosis whose first symptoms of the disease were paroxysmal nausea and vomiting induced by visual perception of movement. Closure of his eyes or cessation of the movements led to a remarkably abrupt termination of symptoms.

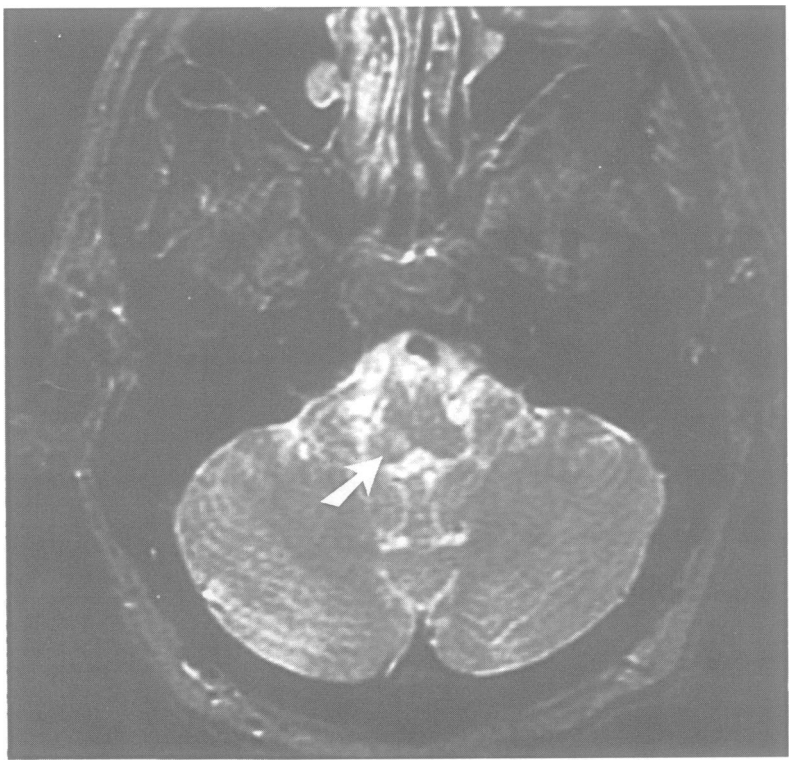
A 47 year old man with the diagnosis of multiple sclerosis was seen in our clinic in August 1993 because of severe paroxysmal nausea and vomiting. These symptoms were induced by perception of any kind of movement in the patient's field of vision. The symptoms would begin abruptly with intense nausea and if the triggering movements persisted, vomiting would soon follow. Movements of any kind (people walking, watching a person getting up from a chair) would all lead to these symptoms. Interestingly, these would occur even if the patient was standing still or lying down in bed. Closure of his eyes or the cessation of movements would abruptly terminate symptoms. These symptoms occurred paroxysmally, lasted three to four hours, and remitted spontaneously. They dated to 1968 when he was first diagnosed with probable multiple sclerosis based on the paroxysmal symptoms and clinical examination. Neurological examination then had shown horizontal gaze evoked nystagmus, generalised hyperreflexia, and bilateral extensor plantar responses. A careful review of the patient's history and records showed paroxysmal nausea and vomiting as the initial manifestations of his disease. Subsequently he had had several such episodes besides other exacerbations including cerebellar ataxia, paraesthesiae and optic neuritis, leading to the diagnosis of clinically definite multiple sclerosis in 1984.

Treatment with routine antiemetics had always failed and over the course of years, the patient had learned to control his symptoms by closing his eyes or having the inciting movements stopped if possible. This turned out to be a consistent cure for his symptoms although they clearly affected his professional and social life. The patient's medical history was otherwise unremarkable.

General physical examination and review of systems including the gastrointestinal system were normal. Neurological examination showed bilaterally decreased olfaction, decreased gustation over the entire tongue, a pale right optic disc, generalised hyperreflexia with extensor plantar responses, horizontal lateral gaze nystagmus, and moderate impairment of tandem walking. During the course of examination, the patient experienced severe nausea, which he attributed to the examiner's movements. He subsequently vomited and then closed his eyes, which led to cessation of nausea and vomiting. Resumption of the examination led to their recurrence and this time, on the patient's request, the examiner remained stationary in his seat, which also resulted in the resolution of symptoms.

Routine serology was negative including rapid plasma reagin, angiotensin converting enzyme, and Lyme titres. Analysis of CSF showed oligoclonal bands with total protein of 67 mg/dl (normal 15–45 mg/dl), but normal cell count and myelin basic protein concentration. Cultures of CSF were negative. An ECG showed sinus bradycardia at a rate of 50/minute; EEG was normal. Auditory and somatosensory evoked potentials were normal. Pattern visual evoked potentials were abnormal on the right, suggestive of a lesion anterior to the optic chiasm, and normal on the left. Brain MRI showed multiple areas of high signal on the T2 weighted images seen in the left optic radiation and throughout the posterior portion of the midbrain and pons, near the collicular plate, and the floor of the fourth ventricle near the area postrema (figure). Cervical MRI was normal.

Paroxysmal symptoms have been reported as the initial manifestations of multiple sclerosis.[2] Vomiting has been reported as a prominent symptom in the disease, especially in the newly diagnosed adolescent population in the early stages.[3] We report paroxysmal nausea and vomiting induced by visual perception of movement as presenting symptoms of multiple sclerosis. These symptoms could be abruptly terminated by cessation of movements or closure of eyes. This was the easiest and



Axial second echo of T2 (TR/TE = 2000/85) shows high signal in the right area postrema consistent with a multiple sclerosis plaque (arrow). (Signa 1·5 T GE Medical Systems, Milwaukee, WI, USA.)