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

G YOUNG
P HUMPHREY
The Walton Centre for Neurology and Neurosurgery, Liverpool L9 1AE, UK

Correspondence to: Dr P Humphrey.


Headache in lateral medullary infarction

Headache is a frequent, although under-emphasised, symptom of lateral medullary infarction. Headache is absent 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). Median survival 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 4 of the patients.

Headache occurred in 26 patients (76%). It began in association with the neurologic deficit in 13 patients, preceded the onset of stroke by one to five days in 10, and by 15 to 28 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 lateral medullary infarction, which was occipital or cervical in 15 (65%), frontotemporal in two (9%), temporal in one (4%), and affecting the eye to forehead in one (4%). It was biocipital in three patients, and monocular 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 cardiogenic emboli (0/4; 0%). Of the 14 patients with angiographically confirmed lesions of the vertebral artery, 12 had dissection or occlusion, in contrast patients (atherothrombotic six, dissection one, fibromuscular dysplasia one) and severe hypotension in six, 13 experienced headache. Headaches 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 ischemia. The underlying cause of headache in cerebral infarction is unknown. Various theories have been proposed in the past, including mechanical compression of 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.

We suggest that headache in lateral medullary infarction is related to thrombus formation in the vertebral artery may suprain the posterior inferior cerebellar artery. 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 (atherosclerosis, severe hypotension, 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 may have two components; one engendered by the process occurring in the artery, the other the result of lesion 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 headache, 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 is the clinical sign of cerebellar infarction especially in the atherothrombotic subgroup.