

## Ischaemic stroke with headache as its only manifestation

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### Abstract

We present two cases of middle-aged men with chronic hypertension presenting with acute severe hemicranial headache with otherwise a normal neurological examination. Investigation revealed occlusion of the ipsilateral middle cerebral artery. We reviewed the literature of ischaemic strokes with headache as the only manifestation and elaborated on the pathophysiology of headaches in ischaemic strokes.

Headache is frequently associated with cerebrovascular disease.<sup>1</sup> Cerebral haemorrhage can cause headache by increasing intracranial pressure, while ischaemic cerebrovascular disease causes headache by disruption of intracranial vessel walls leading to seepage of neurotransmitters and stimulation of receptors on sensory nerve terminals. Infratentorial infarcts are reported to produce more headaches than supratentorial lesions because of the dense sensory innervations of the posterior cerebral vessels.<sup>2</sup>

We present two cases of middle-aged patients presenting with severe periorbital and hemi-cranial headaches with otherwise normal neurologic examination. Investigation revealed middle cerebral artery thrombosis with large territorial infarcts.

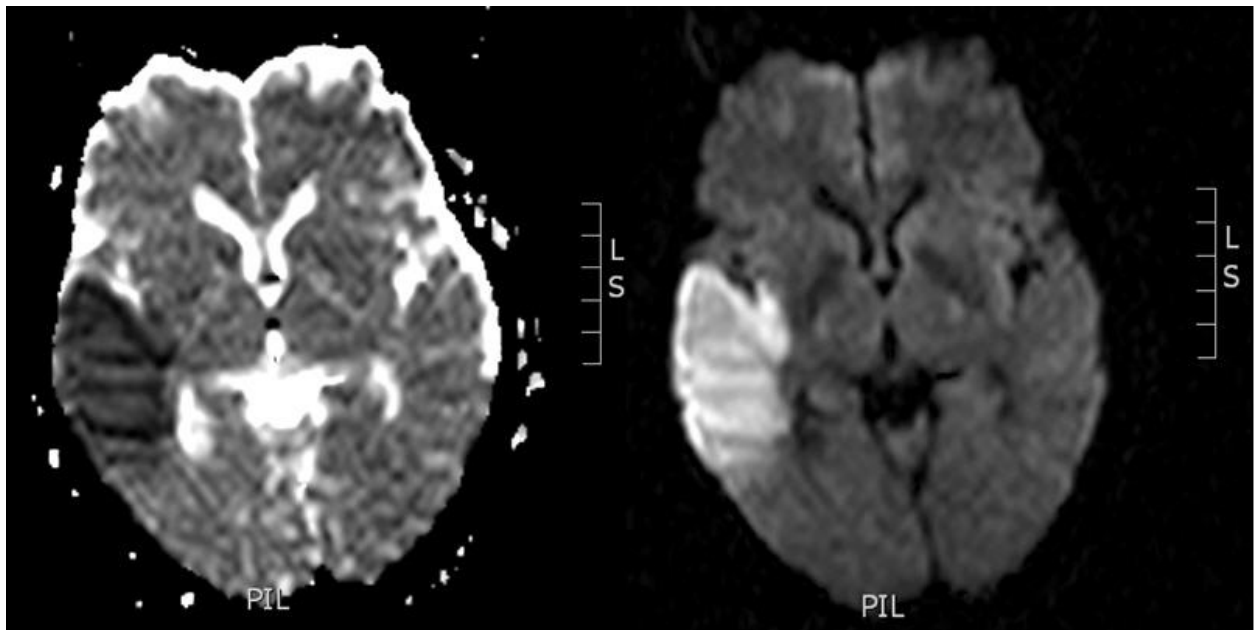
We attempt to explain the pathophysiology of isolated headaches in ischaemic cerebral disease.

### Case reports

**Case 1**—A 53-year-old man with controlled hypertension presented with acute severe persistent right hemicranial headache associated with nausea. No previous history of headaches or other complaints in the days prior to presentation. Blood pressure was 140/85. Completely normal neurological examination. Headache did not resolve on simple analgesics. MRI of the brain revealed large acute infarct in the right temporal lobe. MRA revealed occlusion of the distal branches of the right middle cerebral artery (MCA) with patency of the other cerebral and carotid arteries. The patient was treated with heparin, fearing thromboembolism or paroxysmal atrial fibrillation, followed by anti-platelet therapy with eventual resolution of the headache.

**Case 2**—A 60-year-old man with controlled hypertension presented with acute severe persistent right periorbital pain associated with nausea and vomiting. No previous history of headaches or other complaints in the days prior to presentation. Normal vital signs and full neurological examination. MRI revealed acute infarction in the vascular territory of the right MCA (Figure 1). Cerebral angiography revealed filling defect in the distal branches of the right MCA and a thrombus in the common carotid artery which was the source of the embolus. The patient was treated with heparin fearing thromboembolism or paroxysmal atrial fibrillation, followed by anti-platelet therapy with eventual resolution of the headache.

**Figure 1. Diffusion and apparent diffusion coefficient (ADC) map revealing an acute infarct in the territory of the right middle cerebral artery**



## Discussion

Headache can be a manifestation of an ischaemic cerebral lesion, together with the corresponding neurological deficits, in about 34% of cases.<sup>1</sup> On the other hand, isolated and severe headaches, with no neurologic deficits, associated with MCA infarctions have rarely been reported in the literature.<sup>3</sup>

Headaches may be associated with infarcts of the posterior circulation because of the dense sensory innervations of the posterior cerebral vessels by the trigeminovascular system.<sup>2,3</sup> Supratentorial vessels are sparsely innervated by sensory and autonomic fibres of the trigeminal nerve and thus lesions in these vascular territories usually present with focal neurological deficits rather than headaches. The occasional patient who presents with acute severe headache associated with an MCA infarction confirms the innervation of supratentorial arteries with sensory nerve terminals.

Cerebral arteries are innervated by the trigeminovascular system. The trigeminal nerve terminals are responsible for vascular nociception. The sympathetic fibres induce vasoconstriction and the parasympathetic fibres vasodilatation.<sup>4</sup> Pathological studies have confirmed that arteries and arterioles of the brain are enclosed by a plexus of adrenergic nerves which are superimposed on the media and covered by the adventitia.<sup>5</sup>

The pathology behind headaches secondary to ischaemic strokes is either secondary to mechanical stretching of the thrombotic artery, as described in the mechanism of post endarterectomy headache,<sup>6</sup> or secondary to the release of vasoactive neuropeptides, and the potent vasodilator calcitonin gene related peptide, from the sensory fibres of

the trigeminovascular system. The release of these neuropeptides causes the resulting increased nociceptive input into the nervous system.<sup>3,7</sup>

The vascular theory that claims that headache is secondary to cerebral tissue infarction with corresponding thrombosis of the vasa nervosum seems less probable considering that a minor proportion of patients with infarcts present with severe headaches and that the severity of the headache is not proportionate to the size of the infarct and paranchymal damage.<sup>2</sup>

The headache associated with ischaemic cerebral disease is usually frontal and periorbital because the intracranial vessels are innervated by the ophthalmic ramus of the first branch of the trigeminal nerve that arises from unipolar neurons located in the trigeminal ganglion.<sup>7</sup>

We conclude that headache as the only manifestation of cerebral infarction is not rare and can be seen in patients with supratentorial vessel thrombosis. The reason for the headache is most probably the release of vasoactive neuropeptides such as calcitonin gene related peptide. We recommend MRI rather than CT scan imaging of the brain in patients who present with unexplained, isolated, severe hemicranial or periorbital headaches, especially in patients with increased risk for vascular disease and no explanation for their headache.

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