Review Article

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Headache and Stroke: A Review

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Abstract

Headache is a common disorder that is usually unaccompanied by focal neurological dysfunction. Less commonly, headache may be associated with central nervous system diseases such as stroke. Headache can occur at the time of stroke onset, but it may also precede or follow the onset of stroke. Nevertheless, headaches associated with stroke have not been sufficiently studied, in part because stroke physicians are primarily focused on diagnosis, risk factors, pathophysiological mechanisms, imaging findings, and treatment (e.g., thrombolysis and endovascular therapy) rather than on headache. In this narrative review, I describe the frequency and characteristics of headache across various stroke subtypes, including ischemic stroke, transient ischemic attack, intracerebral hemorrhage, subarachnoid hemorrhage, and other miscellaneous conditions such as venous infarction, arterial dissection, and reversible cerebral vasoconstriction syndrome.

Keywords: Headache, Migraine, Stroke

INTRODUCTION

Headache is a common disorder. The two most common causes of headache are migraine and tension-type headache, and the vast majority of patients with these conditions do not show permanent neurological dysfunction or significant brain abnormality in computed tomography or magnetic resonance imaging (MRI). Less commonly, headache can be caused by head/neck trauma, central nervous system infection, cranial nerve or muscle diseases associated with a variety of causes. Undoubtedly, stroke is one of the etiologies of headache. However, headache is not a usual or dominant symptom of stroke, especially when there are no associated neurological dysfunctions. Exceptions are arterial dissection, venous stroke, reversible cerebral vasoconstriction syndrome (RCVS), or strokes associated with meningitis.

Headaches may precede, occur concomitantly or after the onset of stroke. The reported frequency of headache in stroke patients ranges from 7% to 65%. The extremely wide variation may be due to different enrollment criteria, and different degree of headache evaluation or inability of the headache assessment in patients with certain neurological dysfunction (e.g., altered consciousness, aphasia, dysarthria, etc.).[1] Most importantly, assessment of headache is often neglected in patients with acute stroke when changing neurological dysfunction associated with stroke per se is the primary interest for clinicians.

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HEADACHES ASSOCIATED WITH VARIOUS SUBTYPES OF STROKE

1. Ischemic stroke

Among the patients with stroke who reported the presence of headache by the prospective interview from day 1 to day 8, 86% experienced headache on the day of stroke symptoms, and the remainder had headache at 2–5 days.[2] In another study, 31% had headache prior to, 11% simultaneous with, and 45% after the onset of stroke symptoms.[3] A recent meta-analysis reported that most patients experience headache symptoms on the day of stroke presentation.[1] The headache may disappear within days but can persist for months, or even years.[1]

In most cases (50%–80%), post-stroke headaches manifest as 'tension-type headache' with various descriptions such as pressure, aching or soreness. Symptoms of migraine, e.g., throbbing pain, photophobia, nausea/vomiting are uncommon.[3] The intensity of headache varies from patients to patients. Some studies showed that younger patients more often suffer from post-stroke headache,[4,5] whereas this argument was not supported by another study.[6] In general, female patients complain of headache more often than male patients.

Regarding the location of stroke, some studies reported that the incidence and the intensity of post-stroke headache may be associated with lesions affecting the insular cortex or somatosensory cortical brain. One study[7] used MRI lesion mapping to compare patients with and without headache in ischemic stroke. Authors identified the insular cortex as the region of maximal lesion overlap in those with stroke-related headache. As the insular cortex is a well-established region in pain processing, the results suggest that, at least in a subgroup of patients, acute stroke-related headache might be centrally driven.

In general, cortical strokes are more closely associated with headache than deep, subcortical lacunar infarcts.[1] Studies have also shown that strokes occurring in the posterior circulation, especially in the vertebra-basilar territory develop headache approximately two times more often than those affecting the anterior circulation.[1] There are several putative explanations for this observation that include a difference in trigeminal and autonomic innervation between anterior versus posterior cerebral vessels. Indeed, differences in the innervation pattern of the meninges overlying the posterior cerebral cortices and cerebellum raise the possibility that strokes involving these areas may have a greater chance to stimulate pain-sensing trigeminal fibers. The posterior circulation may also have differential cerebral auto-regulation and become more susceptible to fluctuations in vasomotor tone and permeability. Finally, it is possible that this observation may be related to a higher proportion of migraineurs in the subpopulation of patients with posterior circulation strokes as compared to those with anterior circulation strokes.[1]

1) Thrombotic arterial disease

Patients with ischemic stroke due to large artery atherothrombosis (LAD) often develop headache, that may be explained by dilation of the collaterals or distension of meningeal arteries or trigeminal nerves. Headache may be one of the main predictors for the stroke progression in these patients.[8]

2) Embolic disease

Embolic occlusion at the proximal part of the middle cerebral artery (MCA) or the top of the basilar artery may develop headache associated with corresponding neurological dysfunction. The headache may be caused by distension of the recipient arteries and dilatation of the collaterals.

3) Small vessel disease

Cerebral small vessel disease (SVD) is not associated with atherosclerosis. Rather, lipohyalinosis, fibrinoid degenerations are closely associated. The resultant changes in brain include white matter hyperintensities, microbleeds, lacunar infarction, or perivascular enlargement. Although SVD is associated with neurologic dysfunction such as lacunar syndromes as well as cognitive impairment, they less likely result in headache as compared to strokes associated with LAD or cardiac embolism.

However, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) is an exception. Migraine is a common symptom in patients with CADASIL, and has usually been shown to be the first neurological symptom.[9] In one review paper, 35% of the migraine patients with CADASIL reported symptoms of aura.[10] It seems that genetic abnormalities may be

related with both migraine and CADASIL, although there are arguments against the link between the subcortical nature of lacunar stroke and the occurrence of cortical migraine aura. Nevertheless, cortical spreading depression was found to be enhanced in CADASIL mouse model, and vascular NOTCH3 mutations may increase the susceptibility for spreading depression in CADASIL patients. Some suggest that there are reduced numbers of endothelial progenitor cells and endothelial vascular reactivity in both migraine and CADASIL patients suggesting that certain endothelial dysfunction may link between the two conditions.[8,11] The prevalence of migraine was reported to be higher in European (approximately 43%) than in Asian CADASIL patients (approximately 5%), suggesting the presence of different ethnic susceptibility.[9]

4) Transient ischemic attack

It has been shown that headache occurs in approximately 30% of transient ischemic attack (TIA) patients.[12] Considering the absence of a significant brain damage, it is difficult to understand the pathogenic mechanism of headache in these patients. However, ischemic brain damage, although transient and not readily detected by conventional imaging techniques, may be related to the development of headache. It also has been reported that headaches more often occur in patients with TIA occurring in the vertebrobasilar territory than in those involving the anterior circulation,[12] possibly due to the richer trigeminal innervation in the vertebrobasilar circulation.

2. Hemorrhagic stroke

1) Intracerebral hemorrhage

In general, headache is more common and severe in patients with intracerebral hemorrhage (ICH) than in those with ischemic stroke, probably because of the more severe mass effect that affects the pain-sensitive meninges and trigeminal fibers. Headaches were reported to occur in approximately 60% of ICH patients in the initial stage.[13] It is usually ipsilateral to the hemorrhage, but can be generalized when patients develop increased intracranial pressure and/or hydrocephalus. One study showed that location of the lesion (cerebellar, lobar), female sex, transtentorial herniation are factors associated with headache in ICH patients.[13]

2) Subarachnoid hemorrhage

Headache is the most important symptom of subarachnoid hemorrhage (SAH), that has traditionally been described as 'the worst headache in my life'. However, this expression does not necessarily indicate the presence of SAH because such symptoms may be described in patients with other conditions such as migraine, RCVS or thunderclap headache. Headache is usually maximal at onset of SAH, although some patients may have had one or more preceding headaches (sentinel headache).

3. Arterial dissection

Headache is a notable symptom associated with cerebral arterial dissection. When the affected artery is the extracranial internal carotid artery, the pain is usually located in the neck, often radiating to the ipsilateral face. When a dissection affects the extracranial vertebral artery (VA), the pain usually occurs at the ipsilateral mastoid bone and radiates to the occiput. Pain usually improves over a period of 3–6 months.

For intracranial arterial dissection, pain occurs in 90% of the cases. The pain is usually located to the ipsilateral temporal area in patients with MCA dissection, whereas posterior cerebral artery dissection is associated with ipsilateral eye or occipital pain. Basilar artery dissection is associated with pain in the area of occipital, vertex or diffuse area. With development of advanced imaging techniques such as high resolution vessel wall MRI, dissections occurring in other smaller vessels such as anterior cerebral artery, posterior inferior cerebral artery are occasionally observed, but the location and intensities of pain in these conditions are not properly investigated.

Partly because VA is one of the most frequent sites for dissection,[14] and partly because associated neurological symptoms/signs may be minimal or nonspecific (e.g., dizziness, ataxia, focal facial sensory dysfunction) without significant motor dysfunction, the presence of VA dissection needs to be suspected in patients with sudden neck/occipital pain especially when the symptoms are associated with dizziness or ataxia.

4. Venous stroke

Occlusion of one or several cerebral veins may lead to venous infarction, hemorrhage, or both. This is an uncommon condition, but partly because the symptoms are different from classical arterial stroke and partly because the patients are usually younger than those with usual stroke, the possibility of venous stroke needs to be considered especially when a history taking reveals certain vulnerable conditions: using birth control pills, the postpartum status, head trauma, certain drug ingestion, immunologic disorders such as systemic lupus erythematosis, Bechet's disease, ulcerative colitis, etc., hematologic diseases and infectious disease.

According to the location of venous occlusion, the clinical features vary greatly that include proptosis, conjunctival injection, facial edema, visual disturbances, ophthalmoplegia, hemiparesis etc. Regardless of the location of the venous occlusion, headache is a common symptom occurring in about 70%–80% of the patients. Although there are exceptions, the onset of headache is not as abrupt as SAH. Rather, it increases progressively for days or weeks. Patients are usually treated with anticoagulation. When there is a rapid progression despite the anticoagulation, endovascular therapy can be initiated, which often dramatically improve the patients' clinical symptoms including headache.

5. Reversible cerebral vasoconstriction syndrome

In the 1970s, several women patients were reported who during pregnancy or early puerperium, developed sudden severe headache, nausea, vomiting, seizures and focal neurological dysfunction, that recovered spontaneously within a few weeks. Cerebral angiogram showed arterial stenosis or irregularities that were reversible on repeated angiograms. This was initially called 'postpartum angiopathy'. Similar cases were also reported in patients with migraine or aneurysm. Boston physicians led by Miller Fisher noted the similarity of these cases, and collectively called this as 'Reversible cerebral arterial segmental vasoconstriction syndrome (RCVS)'.

RCVS predominantly affects women, and many are associated with pregnancy and puerperium. It may be associated with the use of vasoactive ergot derivatives. Symptoms may begin during delivery or during 1–2 weeks thereafter. Some patients may develop RCVS at the time of menopause. The headaches are usually generalized but can be localized to the occiput or vertex, and may be exacerbated by physical exertion, straining or coughing. Seizures may occur probably due to brain ischemia or hemorrhages associated with vasoconstriction. Neurological deficits may vary according to the location and extensiveness of brain ischemia or hemorrhages, i.e., motor/sensory dysfunction, dysarthria, aphasia, visual field defect, confusion, and usually improve within several weeks or months. Aneurysmal rupture, vasculitis, arterial dissections should be appropriately differentiated. Although the symptoms and angiographic abnormalities improve spontaneously, vasodilators (calcium channel blockers) with or without steroids are often used. RCVS can also be shown in patients with migraine.

6. Headaches associated with arterial revascularization

Carotid endarterectomy or angioplasty with or without stenting may be associated with headaches that usually begin within hours or weeks after the procedure. The intensity of headache varies from patient to patient. The headache may be accompanied by focal neurological deficits or seizures that are usually associated with newly developed brain lesions. The headache appears to be caused by sudden reperfusion (increased cerebral flow) and resultant severe brain edema or hemorrhage. Brain MRI demonstrates cerebral edema, hemorrhages or infarction. Patients usually show markedly increased blood pressure, and aggressive blood pressure reduction is needed to prevent and treat this so-called reperfusion syndrome.[15]

MIGRAINE AND PATENT FORAMEN OVALE

Although not fully explained, the patent foramen ovale (PFO) is suggested to play a role in the pathophysiology of migraine. Proposed mechanisms include the passage of micro-emboli, metabolites, and vasoactive substances through the PFO, and transient hypoxemia that results in micro-infarcts in the brain.[16] Nevertheless, the association between migraine and PFO has been questioned by previous studies.[17] Some randomized controlled trials (RCTs)

showed that PFO closure was associated with a significant reduction in the number of migraine attacks. However, complete resolution of migraine was only demonstrated by observational studies and not by RCTs. These results seem to provide insufficient support for PFO closure exclusively to treat migraine, although reduction of migraine symptoms may be observed in some patients. This notion is supported by recent comprehensive reviews,[18] which emphasized the need for additional evidence from RCTs. Instead, the guideline-recommended use of monoclonal antibodies targeting the calcitonin gene-related peptide pathway seems to offer another effective pharmacological alternative.[19] Within the context of mixed evidence from PFO closure and alternative pharmacological treatments, PFO closure is not considered as a first-line/routine treatment for migraine.[20]

MOYAMOYA DISEASE

Moyamoya disease (MMD) is a progressive steno-occlusive cerebrovascular disorder of the intracranial internal carotid arteries or proximal middle cerebral arteries characterized by fragile, compensatory collateral vessel formation. Although ischemic and hemorrhagic strokes are the best known clinical manifestations, headaches are relatively common and often disabling.[21] Epidemiological studies have reported that headache occurs in 17%–85% of MMD patients, with particularly high rates among pediatric patients.[22] Headache phenotypes include migraine-like headaches with or without aura, tension-type, cluster, and hemiplegic variants. A relatively large study showed that 44 of 204 children (21.6%) with MMD suffered from headache, and nausea/vomiting were seen in 12. In four, headache developed during hyperventilation, and in three, TIA and headache occurred simultaneously.[23]

These presentations mimic primary headaches, and there can be delays in the diagnosis of underlying MMD. The pathophysiology of MMD-related headaches seems to be multifactorial, involving vascular stenosis, abnormal collateral circulation, altered hemodynamics, and neurogenic inflammation.[21,22] Chronic hypoperfusion may lower the threshold for cortical spreading depression, contributing to migraine-like symptoms. Surgical revascularization may alleviate headaches,[21] but persistent or new headaches may develop postoperatively. Therefore, headaches are more often managed with analgesics or non-steroidal anti-inflammatory drugs. Vasoconstrictive agents (e.g., triptans) are not generally recommended, but non-vasoconstrictive agents are being developed.[22] Further studies are needed to elucidate the epidemiology, pathophysiological mechanisms, and appropriate management of headaches associated with MMD.

CONCLUSION

Headaches may precede, occur concomitantly, or after the onset of stroke. However, in the majority of the patients, headache is not a major symptom of stroke. Therefore, headache has not gained sufficient attention among stroke neurologists. In the clinical practice, careful observation of headaches may help us understand the etiologies of strokes especially SAH, arterial dissection, venous stroke, RCVS, or meningitis. It may also give us a clue for the progress of stroke. For instance, increasing intensity of headache suggests the presence of mass effect, increased ICP, and potential herniation, and may prompt us to detect and treat stroke cases appropriately. Headaches occurring in patients with CADASIL or PFO may allow us to understand the relationship between the two seemingly unrelated condition, migraine and cerebral ischemia. Further studies are needed that will eventually allow us to understand such theoretically and practically important issues in patients with cerebrovascular diseases.

AVAILABILITY OF DATA AND MATERIAL

The data presented in this study are available upon reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

Conceptualization: JSK; Writing–original draft: JSK; Writing–review & editing: JSK.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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