

Migrainous Infarction Involving Two Different Arterial Territories: Report of Two Cases

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Abstract- It is reasonable to speculate that migrainous infarction may develop in different territories of cerebral arteries. However, this condition was rarely reported before. Here we described two patients (one woman, 29 year-old; one man, 47 year-old) with a history of migraine with aura, had ischemic strokes during the migraine attack. The ischemic stroke of the female patient involved the territories of the posterior cerebral and anterior choroidal arteries; whereas the male patient had two episodes of ischemic strokes, involving the territories of the middle and posterior cerebral arteries sequentially. Both patients recovered well during the follow-up periods. We suggest that migrainous infarction can involve different arterial territories.

Key Words: Migraine, Migrainous infarction, Cortical spreading depression

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INTRODUCTION

Migraine with aura has been known as a risk factor for ischemic stroke, particularly in the young adults⁽¹⁾. Previous investigations have shown some characteristics of migrainous infarction, such as young female predominance, and a more frequent involvement of the posterior circulation than the anterior circulation. The involved arteries in migrainous infarction may be occlusion or diffuse narrowing due to vasospasm^(2,3). However, migrainous infarction occurring in different territories of cerebral arteries was rarely reported before^(4,5). Here we presented two cases of migrainous infarction: one patient cannot be attributed to single arterial territory

and the other had two episodes of ischemic strokes involving different arterial territories sequentially.

CASE HISTORY

Patient 1. A 29-year-old woman was diagnosed to have migraine with aura since age 12. The manifestation of her migraine was intermittent unilateral throbbing headache preceded by transient binocular blurred vision. Her medical history was unremarkable for diabetes mellitus, hypertension, heart disease or smoking. Besides, she did not use oral contraceptives or any illicit drugs. She had neither family history of migraine nor cerebrovascular diseases. Three days before the admis-

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sion, she suffered from acute onset of blurred vision, unsteady gait, and weakness and numbness of the left extremities, followed by severe throbbing headache on the left temporal area. Blurred vision recovered gradually, but unsteady gait, left hemiparesis, and hemiparesthesia persisted. Fluid-attenuated inversion recovery (FLAIR) images of the brain MRI showed two high signal intensity lesions at the right lateral thalamus, the posterior limb of the internal capsule and the medial occipital areas (Fig. 1A-B). The former lesion was regarded as the anterior choroidal artery territory from the clinical presentation of hemiparesis and hemiparesthesia and typical location on MRI and the other one was contributed to the posterior cerebral artery territory. Series of studies including serum autoimmune and coagulation profile (protein C, protein S, antithrombin III, and anticardiolipin antibody), cerebrospinal fluid study, carotid

duplex, and transcranial Doppler ultrasound, echocardiography, and cerebral angiography were normal. Therefore, migrainous infarction involving the right posterior cerebral and anterior choroidal arteries was diagnosed. Her neurological deficits improved gradually and only mild weakness of the left extremities left after discharge.

Patient 2. A 47-year-old man had intermittent transient visual loss or scintillating scotoma in the left visual field, followed by unilateral throbbing headache since adolescence. Migraine with aura was diagnosed. He did not have systemic diseases, or the habit of smoking. His family history of migraine was negative. One week before the admission, acute onset of numbness and clumsiness of the left extremities developed and followed by throbbing headache on the right temporal area. Headache subsided gradually but left side numbness and

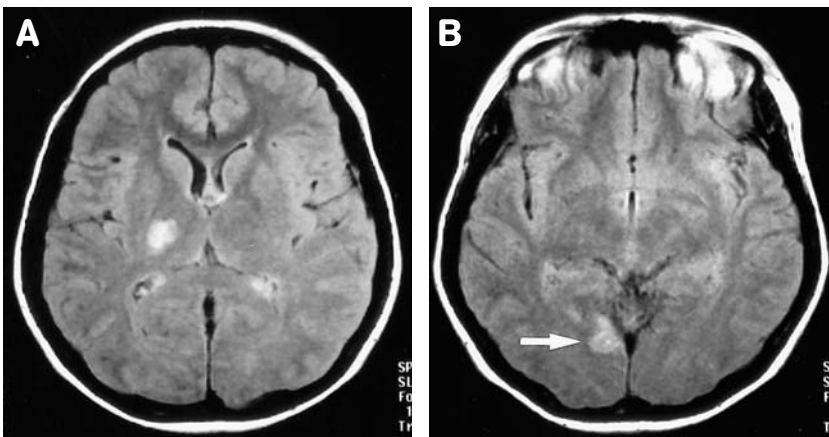


Figure 1. FLAIR images of the head MRI showed two high signal intensity lesions at the right thalamus, the posterior limb of the internal capsule (A) and the medial occipital cortical and subcortical areas (B)(arrow).

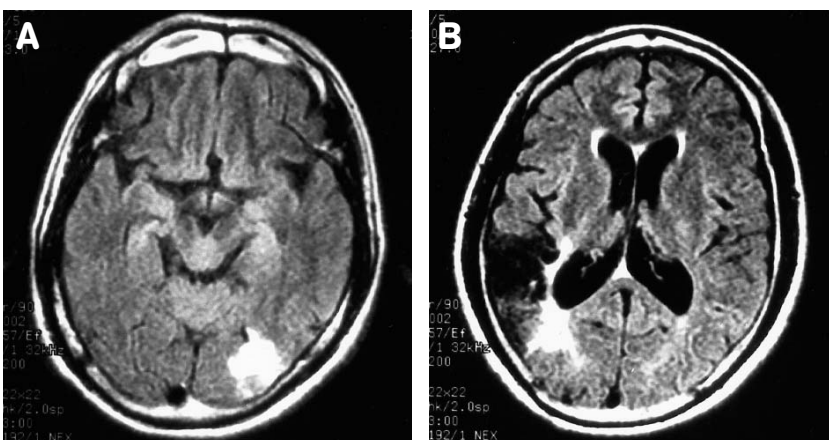


Figure 2. FLAIR images of the head MRI showed a wedge shape lesion with high signal intensity at the left occipital lobe (A), and an old insult with negative mass effect at the right temporo-parietal lobes (B).

mild weakness persisted. T2 weighted images of the brain MRI showed one high signal intensity lesion at the right temporo-parietal areas attributed to the middle cerebral artery territory. Series of work-up including serum autoimmune and coagulation profiles, carotid duplex, and trans-thoracic and trans-esophageal echocardiography were all within normal limits. Cerebrospinal fluid study and cerebral angiography were not performed because of the patient's declination. Low dose aspirin was prescribed for stroke prevention. However, 4 years later, he suffered from acute onset and persistent right visual field defect with coexistent throbbing headache. Except for the right homonymous hemianopsia, no other neurological deficits were found in our clinic. The FLAIR images of the brain MRI showed a wedge shape lesion with high signal intensity at the left occipital lobe (Fig. 2A). Previous old insult at the right temporo-parietal lobes without mass effect was also noted (Fig. 2B). MR angiography of the circle of Willis appeared no definite abnormality. Therefore, recurrent ischemic strokes related to migrainous infarction involving the left posterior cerebral artery territory was diagnosed. The patient recovered well at the 3 years follow-up.

DISCUSSION

Our two cases fulfilled the definition of migrainous infarction as proposed by the Headache Classification of the International Headache Society^(6,7). They had a history of migraine with aura and their strokes occurred during the course of typical migraine attack. Both patients had no conventional cardiovascular risk factors, and series of work-up did not disclose other causes of stroke. Besides, conventional or MR angiography didn't detect vascular abnormality. The interesting findings in our patients were that one patient had cerebral infarct simultaneously involving territories of the posterior cerebral and anterior choroidal arteries and the other patient had two episodes of cerebral infarct involving territories of the middle cerebral and posterior cerebral arteries sequentially.

From the literature review, migrainous infarction involving two vascular territories concurrently has been reported only in a few cases. One case report described a

young migraine sufferer who had bilateral infarcts of the posterior cerebral arteries during the course of his classic migraine⁽⁴⁾. Another case report showed a migrainous infarction involving bilateral anterior cerebral artery territories⁽⁵⁾. In a report of 20 cases of migrainous infarction by Broderick and Swanson's⁽⁸⁾, four cases probably occurred more than one arterial territory, but the clinical information was not described in detail. To our knowledge, migrainous infarction involving the posterior cerebral and anterior choroidal arteries concurrently as in our case 1 has never been reported.

Numerous hypotheses have been raised to explain the pathogenesis of migrainous infarction^(2,3,7). Prolonged migraine aura induced regional cerebral ischemia is one of the important theories. Migraine aura has been known to be caused by the cortical spreading depression⁽⁹⁾, which is a depolarization wave that propagates slowly across the brain cortex and is associated with transient depression of neuronal activity. The cerebral blood flow changes during migraine aura are secondary to the depressed neuronal activity, thus the area of oligemia might not match single arterial territory⁽¹⁰⁾. In one case report, cerebral hypoperfusion occurred in bilateral occipital regions and slowly progressed anteriorly during the migraine aura was demonstrated directly by positron emission tomography study⁽¹¹⁾.

From this aspect, we propose that migrainous infarction involving two different vascular territories concurrently or sequentially, as our presented cases are reasonable. However, this point was not emphasized before. Therefore, some cases of migrainous infarction may be misclassified as an embolic stroke or other etiologies rather than stroke just because more than one vascular territory is involved.

In conclusion, our report suggests that migrainous infarction may develop in two different arterial territories. This phenomenon also correlated well with one of the possible mechanisms of migrainous infarction.

REFERENCES

1. Rothrock J, North J, Madden K, et al. Migraine and migrainous stroke: risk factors and prognosis. *Neurology* 1993;43:2473-6.

2. Caplan LR. Migraine and vertebrobasilar ischemia. *Neurology* 1991;41:55-61.
3. Hoekstra-van Dalen RA, Cillessen JP, Kappelle LJ, et al. Cerebral infarcts associated with migraine: clinical features, risk factors and follow-up. *J Neurol* 1996;243:511-5.
4. Moen M, Levine SR, Newman DS, et al. Bilateral posterior cerebral artery strokes in a young migraine sufferer. *Stroke* 1988;19:525-8.
5. Demirkaya S, Odabasi Z, Gokcil Z, et al. Migrainous stroke causing bilateral anterior cerebral artery territory infarction. *Headache* 1999;39:513-6.
6. Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1988;8(Suppl 7):1-96.
7. Welch KM, Levine SR. Migraine-related stroke in the context of the International Headache Society classification of head pain. *Arch Neurol* 1990;47:458-62.
8. Broderick JP, Swanson JW. Migraine related strokes. *Arch Neurol* 1987;44:868-71.
9. Ferrari MD. Migraine. *Lancet* 1998;351:1043-51.
10. Lauritzen M. Pathophysiology of the migraine aura: the spreading depression theory. *Brain* 1994;117:199-210.
11. Woods RP, Iacoboni M, Mazziotta JC. Bilateral spreading cortical hypoperfusion during spontaneous migraine headache. *N Engl J Med* 1994;331:1689-92.