

Symmetrical and Regressive Cerebellar Lesions in a Migrainous Brain: Case Report

Migrenöz Bir Beyinde Simetrik ve Gerileyen Serebellar Lezyonlar

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ABSTRACT Migraine is a widespread, chronic, multifactorial, neurovascular disorder typically characterized by throbbing recurrent headache attacks. Here we report a 38-year-old female patient admitted to emergency room because of sudden onset of vertigo, nausea, vomiting, and headache and diagnosed as migraine with aura and had bilateral and symmetrical lesions in the cerebellum, which showed regression in following weeks. Ischemic lesions can develop in the brain, in the posterior system and especially in the cerebellum of women having migraine with aura accompanied with being a cigarette smoker and/or using oral contraceptive drugs and being under the age of 50 year. Those lesions can be regressive. As a conclusion it can be suggested that migraine may be a risk factor for infarct-like lesions on the posterior circulation area of the brain. The region in which these lesions are seen most commonly is the cerebellum.

Key Words: Migraine disorders; cerebellum

ÖZET Migren tipik zonklayıcı tarzda, rekürren baş ağrısı atakları ile karakterize yaygın, kronik, multifaktöriyel nörovasküler bir bozukluktur. Biz burada, acil servise ani başlangıçlı baş dönmesi, bulantı, kusma ve baş ağrısı ile kabul edilip, auralı migren tanısı alan ve serebellumda bilateral, simetrik ve takibeden haftalarda gerileme gösteren lezyonları olan 38 yaşında bir kadın hasta bildirmekteyiz. Özellikle, 50 yaş altındaki, sigara içen ve/veya oral kontraseptif kullanan, auralı migreni olan kadınlarda, beyinde, posterior sistemde iskemik lezyonlar oluşabilir. Bu lezyonlar gerileyici olabilir. Sonuç olarak öne sürülebilir ki migren, beyin arka dolaşım bölgesindeki infarkt benzeri lezyonlar için bir risk faktörü olabilir. Bu lezyonların en yaygın görüldüğü bölge serebellumdur.

Anahtar Kelimeler: Migren hastalıkları; serebellum

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Migraine is a widespread, chronic, multifactorial, neurovascular disorder typically characterized by throbbing recurrent headache attacks and autonomic nervous system dysfunctions (migraine without aura), accompanied by neurological manifestations (migraine with aura) in one-third of the patients.¹

Migraine appears to be a risk factor for the development of infarct-like lesions, especially involving the cerebellum.²

Using T₂-weighted magnetic resonance imaging, brain white matter abnormalities have been detected in patients with migraine with frequen-

cies ranging from 6% to 46%. Specifically in patients with migraine with aura, in the infra and supratentorial regions, an increased infarct risk has been reported.¹

CASE REPORT

A 38-year-old female patient had been admitted to emergency room of another medical center 10 days prior to her admission to our outpatient clinic, because of sudden onset of vertigo, nausea, vomiting, and headache.

Approximately 2 hours after a symptomatic treatment with nonsteroid analgesics and antiemetics administered there to her, she had had relief from all her symptoms. The neurological and systemic examinations during her complaints had been assessed as unremarkable.

In her history, there had been two additional similar complaints within 10 years, 3 episodes of bright-line visions experienced for nearly 10 minutes occurring generally in both eyes within 3 recent months, and unilateral throbbing headache happening time to time as a result of stressful conditions and lasting nearly 24 hours. Her history also included 2 packages of cigarette smoking per day for 17 years and no systemic disorders were determined.

In her parental medical history, her mother was diagnosed as migraine with aura.

On the FLAIR-weighted images obtained by cranial magnetic resonance imaging (MRI) that was carried out on the subsequent day of the onset of the complaints, bilateral cerebellar hyperintense lesions (Figure 1a) showing diffusion restriction (Figure 1b) suggesting ischemia were observed. According to this observation, the patient was examined with respect to etiology of ischemic cerebrovascular accident.

Carotis and vertebral Doppler USG, cranial computerized angiography, and echocardiography investigations were normal. The results of thyroid function tests, biochemical analysis, vitamin B12, folic acid, homocysteine levels, hemogram, and hemostasis examination were normal. ASO, CRP, RF, immunoglobulin and complement levels, protein C-S, antithrombin III levels, and activated protein

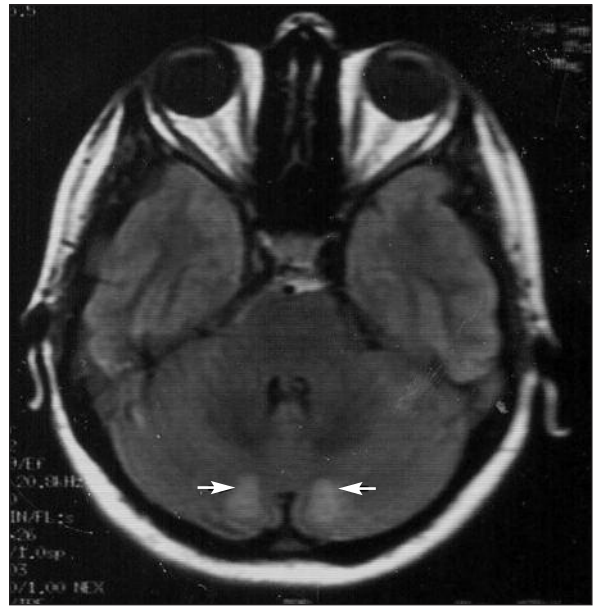


FIGURE 1a: On FLAIR weighted images, bilateral, symmetrical hyperintense lesions were seen in cerebellum at subsequent day of symptoms (arrows show the lesions).

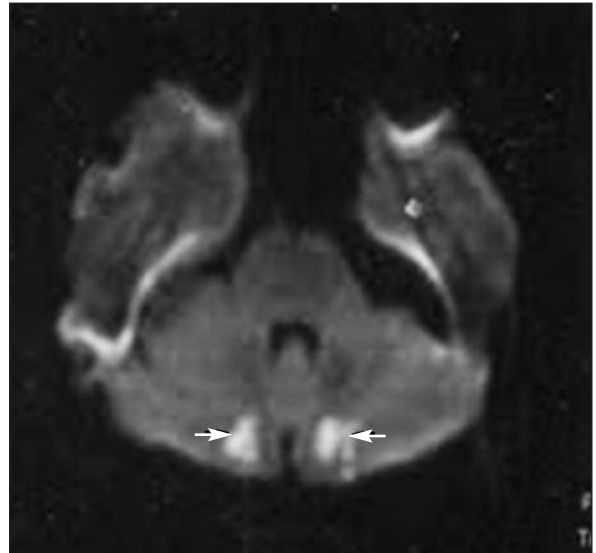


FIGURE 1b: On diffusion weighted images bilateral symmetrical lesions showing diffusion restriction were seen in cerebellum at subsequent day of symptoms (arrows show the lesions).

C resistance were also normal. Factor V Leiden mutation, prothrombin 20210 mutation, and PAI gene mutations were negative.

MTHFR was determined as heterozygote. ANA, anti-ds DNA, p-ANCA, c-ANCA, lupus anti-coagulant, anticardiolipin Ig G and Ig M, and serologic tests for brucellosis were negative.

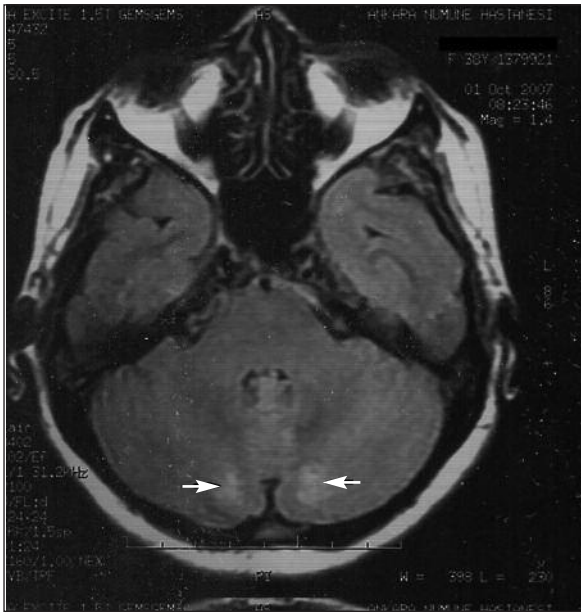


FIGURE 2: Vanishment in size and intensity of the lesions were seen on the cranial MRI FLAIR images one week later (arrows show the lesions).

Cranial MRI FLAIR and T₂-weighted images and the diffusion sequences of the patient, obtained a few weeks later, showed a significant diminishment in lesion size and a significant decrease in signal intensity (Figure 2).

Under the light of current history, medical examination, and laboratory findings, we diagnosed our patient as migraine with aura according to the International Headache Society criteria II (IHS-II).³

Migraine may be related to subclinical, vascular cerebral lesions. Those lesions can be seen by the help of magnetic resonance imaging even without patient having clinical stroke history and the concerned lesions affect especially the posterior vascular system of the brain and more specifically the cerebellum.^{2,4} An association between migraine and cerebellum was reported by clinical and pathophysiological evidence.⁵ In a study by Reinhard et al. altered cerebellar autoregulation was shown in patients with migraine with aura.⁶ In our case, there were lesions consistent with the acute ischemia in bilateral cerebellar hemispheres, obtained by cranial imaging without any stroke history (as supported by the data obtained from the neurological examination performed at the time of event, in spite of the intense nausea-vomiting and

headache). Just like our patient, in the CAMERA study none of the patients with cerebellar lesions appearing as infarcts on MRI had a clinical history of stroke.²

As well as migraine there might have been a predisposition for stroke, while on the other hand stroke, just like migrainous infarct, may be a direct result of migraine itself. Correlation between migraine and stroke is stronger for the stroke subtypes occurring in the under 50-year-old age group and for the ones occurring in female patients. Recent evidences show higher stroke risk factors for migraine with aura than for migraine without aura and again higher risk factors for cigarette smokers than nonsmokers and for female oral contraceptive consumers than non consumers.^{4,7} Our patient was also under 50 years of age, and was a cigarette smoker and had migraine attacks with aura. But she did not have exact posterior system stroke symptoms and the cranial lesions were found co-incidentally. The cranial MRI was performed because of severe nausea and vomiting. MTHFR C677T polymorphism is seen as a candidate condition for susceptibility to migraine. So much that, concerned polymorphism is the most studied predisposition for the cerebral ischemia.⁸ In our case, MTHFR C677T was heterozygote but since folic acid and homocysteine levels were normal we did not consider it as a risk factor.

Cardiac causes such as patent foramen ovale and mitral valve prolapsus increase risk factors for ischemic cerebral lesions for patients with migraine⁹. However in our patient no cardiac risk factor has been determined.

Cranial magnetic resonance images re-obtained a few weeks after the initial symptoms, showed less intensity of lesions and in the size. We have encountered a case report with vanishing cerebellar infarcts reported in the literature¹⁰. As mentioned above population-based studies, which were recently performed, suggest migraine to be a risk factor to the infarct-like lesions on the posterior circulation area.⁹ The region that those infarcts are seen most commonly is the cerebellum.² These lesions are thought to be as real vascular infarcts when

their size, their locations, and their MRI characteristics are taken into account. However, because there are no postmortem studies performed on the pathologies of those MRI findings yet, their etiologies are not known exactly.¹⁰ Due to the regression of our patient's lesions we needed to question ischemic pathology. However, it has been reported that for some real ischemic conditions, abnormalities seen on MRI images can be transient. Diffusion-weighted images can display early ischemic changes and lesion regression may be seen on diffusion-weighted images in some stroke cases.¹¹ In our case as well, diminishing of lesion size was observed after a few weeks. However, observed regression seen on diffusion-weighted images neither defines the real cerebral ischemia, nor excludes it. It is known that,

for patients with migraine, certain abnormalities can be seen in the central nervous system as well as on MR spectroscopy that suggest mitochondrial dysfunctions. The real origin of infarct-like lesions for migraine has not been determined yet.¹² It is also not known whether those lesions are constant or progressive. For rare situations in literature, similar to our case, it has been shown that those lesions can be regressive. As a result, when we assess our case under the light of literature we can conclude that ischemic lesions can develop in the brain, in the posterior system and especially in the cerebellum of women having migraine with aura, being cigarette smokers and/or using oral contraceptive drugs, and being under the age of 50. Those lesions can be bilateral, symmetrical, and regressive and rare.

REFERENCES

1. Degirmenci B, Yaman M, Haktanir A, Albayrak R, Acar M, Yucel A. Cerebral and cerebellar ADC values during a migraine attack. *Neuroradiology* 2007;49(5):419-26.
2. Kruit MC, Launer LJ, Ferrari MD, van Buchem MA. Infarcts in the posterior circulation territory in migraine. The population-based MRI CAMERA study. *Brain* 2005;128(Pt 9):2068-77.
3. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. 2nd ed. *Cephalalgia* 2004;24(Suppl 1):9-160.
4. Sacco S, Cerone D, Carolei A. Comorbid neuropathologies in migraine: an update on cerebrovascular and cardiovascular aspects. *J Headache Pain* 2008;9(4):237-48.
5. Sığırılı D, Özdemir ST, Ercan İ, Taşkapılıoğlu Ö, Hakyemez B, Şener DK, et al. Statistical shape analysis of the corpus callosum and the cerebellum in migraine patients. *Turkiye Klinikleri J Med Sci* 2013;33(1):200-9.
6. Reinhard M, Schork J, Allignol A, Weiller C, Kaube H. Cerebellar and cerebral autoregulation in migraine. *Stroke* 2012;43(4):987-93.
7. MacClellan LR, Giles W, Cole J, Wozniak M, Stern B, Mitchell BD, et al. Probable migraine with visual aura and risk of ischemic stroke: the stroke prevention study. *Stroke* 2007;38(9):2438-45.
8. Pezzini A, Grassi M, Del Zotto E, Giossi A, Monastero R, Dalla Volta G, et al. Migraine mediates the influence of C677T MTHFR genotypes on ischemic stroke risk with a stroke-subtype effect. *Stroke* 2007;38(12):3145-51.
9. Kruit MC, van Buchem MA, Hofman PA, Bakkers JT, Terwindt GM, Ferrari MD, et al. Migraine as risk factor for subclinical brain lesions. *JAMA* 2004;291(4):427-34.
10. Rozen TD. Vanishing cerebellar infarcts in a migraine patient. *Cephalalgia* 2007;27(6):557-60.
11. Schulz UGR, Briley D, Meagher T, Molyneux A, Rothwell PM. Abnormalities on diffusion weighted magnetic resonance imaging performed several weeks after a minor stroke or transient ischemic attack. *J Neurol Neurosurg Psychiatry* 2003;74(6):734-8.
12. Montagna P. Magnetic resonance spectroscopy in migraine. *Cephalalgia* 1995;15(4):323-7.