

# Vertebral Artery Dissection Presented as Lateral Medullary Syndrome in a Patient with Migraine : A Case Report

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## Abstract-

**Purpose:** Migraine and artery dissection are both rare causes of ischemic stroke<sup>1</sup>. The mechanism of migraine-related intracranial artery dissection is still unknown. It is proposed that the repeated attack of migraine would make the involved artery more vulnerable to tearing and lead to dissection<sup>3</sup>.

**Case report:** We describe a 42-year-old female suffering from basilar-type migraine for more than 20 years. The patient complained severe dizziness with hyperventilation while watching television. Initially anxiety and migraine attack were impressed in the emergency room, but dizziness accompanied with dysarthria and dysphagia was noted later. After admission, lateral medullary syndrome was suspected after a detailed neurological examination, and a brain magnetic resonance image (MRI) revealed an acute infarction on the left lateral medulla oblongata, confirming the clinical diagnosis. Furthermore, cerebral angiography revealed the left distal vertebral artery dissection. The patient was reluctant to use an anticoagulant; therefore aspirin was given for secondary stroke prevention and topiramate for migraine prophylaxis.

**Conclusion:** The exact mechanism of migraine-related intracranial artery dissection has yet to be proven, we propose that this may be caused by vessel wall edematous changes with repeated migraine attacks resulting in sudden or unusual stretching.

**Key Words:** migraine, vertebral artery dissection, lateral medullary syndrome, topiramate

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## INTRODUCTION

Migraine and artery dissection are rare causes of ischemic stroke<sup>(1)</sup>. From previous epidemiological studies, migraine patients, especially those with aura had a higher incidence of ischemic stroke<sup>(2)</sup>. The mechanism of migraine related intracranial artery dissection is still

unknown. It is proposed that the repeated attack of migraine would make the involved artery more vulnerable to tearing and lead to dissection<sup>(3)</sup>.

## CASE REPORT

A 42-year-old female suffered from basilar-type

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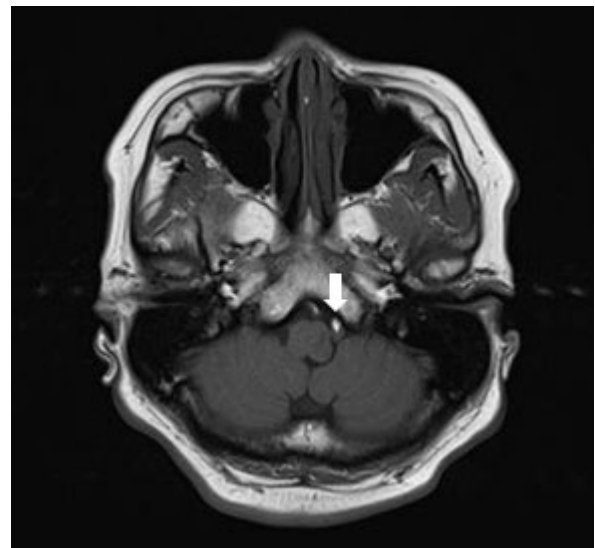
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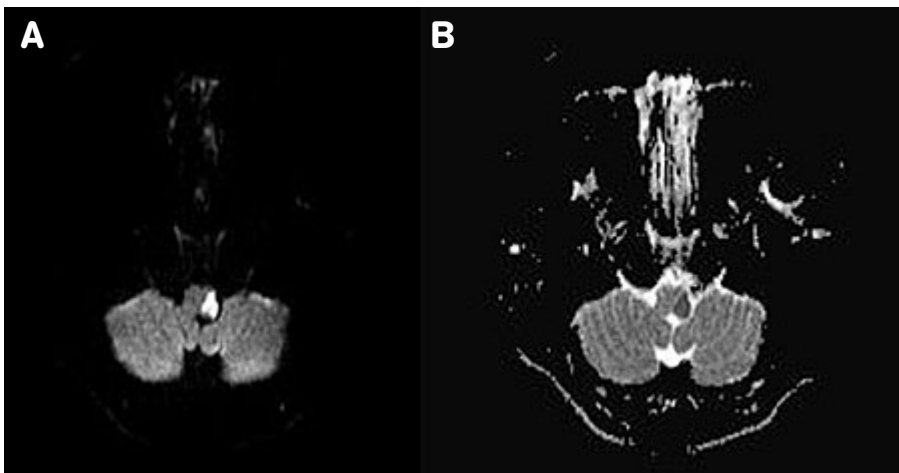
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migraine for more than 20 years without taking any prophylactic medications. The throbbing headache usually happened about 10 to 30 minutes after attacks of vertigo, tinnitus and transient numbness of one side of face, trunk and limbs. Double vision and slurred speech attacked occasionally. These symptoms were more severe during menstrual period. Complaining of severe dizziness, hemi-paresthesia, and hyperventilation while watching television at home, the patient visited the emergency room at around 9 PM. Anxiety with hyperventilation syndrome was initially impressed at the emergency room. Routine blood and biochemistry examinations were normal. The patient had a normal heart rate at around 60 beats per minute; however, blood pressure was elevated to 192/117 mmHg. Intravenous normal saline hydration with nasal cannula oxygen supply was given; however, dizziness and dysarthria persisted, and dysphagia was also noted later. After admission, the neurological examination showed left arm hypotonia with dysmetria, upward jerk-like nystagmus, and ataxia. A loss of pain and temperature sensation in the right limbs and trunks along with left facial paresthesia was also noted. Also, left vocal cord palsy was noted by nasopharyngeal scope examination. Brain computer tomography (CT) taken in the emergency room was negative; however, a brain MRI after admission revealed a hyperintense focus in the left medulla oblongata with restrictive diffusion, which demonstrated high signal on the diffusion weighted image and low on the apparent diffusion coefficient image, consistent with an acute infarct and com-

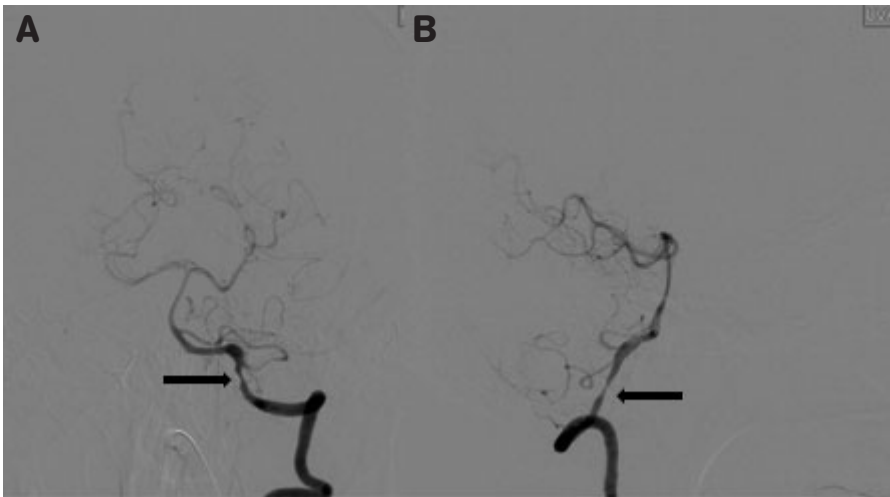
patible with lateral medullary syndrome (Figure 1). Since there was an equivocal eccentric wall thickening in the left vertebral artery on the T1 weighted image (Figure 2), an angiography of the vertebral arteries was performed. In the left vertebral arteriogram, a focal and segmental stenosis was identified in the left distal vertebral artery (VA), proximal to the left posterior inferior cerebellar artery (Figure 3), corresponding with the MRI finding. The diagnosis of a left VA dissection was established. A complete young stroke risk factors survey



**Figure 2.** On this axial T1 weighted image at the level of medulla oblongata, there was an eccentric wall thickening in the left distal vertebral artery (arrow), corresponding to the image findings of vertebral angiography, and it indicated an arterial dissection.



**Figure 1** A small lesion with diffusion restriction was found in the left lateral medulla oblongata with a high signal on diffusion weighted image (A), and low signal on apparent diffusion coefficient image (B).



**Figure 3.** On digital subtraction angiography, an abrupt short segmental narrowing was found in the left distal vertebral artery (arrow) both on the Townes (3a) and lateral views (3b), suggestive of an arterial dissection.

including lipid, autoimmune profile, coagulation factors and cardiac workup were normal. She also refrained from use of oral contraceptive and cigarette in the past. Therefore, we proposed that neurogenic inflammation induced by repeat migraine attack is the major predisposing factor to the cause of non-traumatic arterial wall injury. Aspirin was given for stroke prevention. In addition, topiramate 25mg twice a daily was prescribed to prevent a migraine attack. Patient was discharged smoothly with regular follow-up visits at an out-patient clinic. A computer tomography angiography follow-up 6 weeks later revealed a complete recanalization of the dissected vessels.

## DISCUSSION

The patient we reported in this article had had basilar-type migraine for more than 20 years without regular or prophylactic medications. Her life-style habits were healthy and no significant young stroke risk factors were found. She was presented with left lateral medullary syndrome due to left vertebral artery dissection confirmed by brain magnetic resonance image and angiography. Vertebral artery dissection due to chronic basilar-type migraine is suspected. However, making a direct correlation between vertebral artery dissection and migraine could be problematic.

Basilar-type migraine used to be called basilar artery migraine or basilar migraine and is a rare and complicated migraine with symptoms caused by brainstem dysfunction. Therefore, it is still difficult to be recognized in clinical practice. According to the International Classification of Headache Disorders, 2nd edition (ICHD-2), basilar-type migraine has to have at least 2 attacks that meet the following criteria. It has to fulfill the migraine without aura criteria and no motor weakness. At least 2 of the following reversible neurologic symptoms are present including dysarthria, vertigo, tinnitus, hypacusia, diplopia, homonymous visual symptoms, motor incoordination, consciousness disturbance or bilateral sensory symptoms. At least one symptom occurs gradually over (at least) 5 minutes, and/or consecutive symptoms occur over (at least) 5 minutes. Each symptom has to last over (at least) 5 minutes and at least 60 minutes<sup>(8)</sup>. Serious episodes of basilar-type migraine can cause stroke, coma and even death<sup>(7)</sup>.

A previous epidemiologic study in young women showed that migraine doubles the relative risk of stroke<sup>(2)</sup>. Recent meta-analysis studies conclude that the relative risk (RR) of stroke in migraine patients is 2.16 times higher than non-migraine patients, and the relative risk for migraine patients with aura is higher than those without aura (2.27 vs 1.83)<sup>(4)</sup>. Furthermore, women younger than 45 years old might have a higher relative

**Table 1.** Summary of the case reports on migraine with cervical artery dissection

Author	Age/Sex	Vessel dissected	Migraine with aura
Casto <sup>18</sup>	22/male	LICA	Unknown
Young <sup>19</sup>	35/female	RVA	+
Duyff <sup>20</sup>	36/male	RICA	+
Ganesan <sup>21</sup>	15/male	RICA	-
Mirza <sup>22</sup>	39/male	RICA	Unknown
	27/female	RICA	
Silverman <sup>23</sup>	3 cases	ICA	+
Lotze <sup>24</sup>	8/male	LVA and BA	Unknown
Buttinelli <sup>25</sup>	34/female	LICA	-
Rozen <sup>26</sup>	61/male	LVA	Unknown
Morelli <sup>27</sup>	47/male	RVA	+

LICA left internal carotid artery, RICA right internal carotid artery, LVA left

Vertebral artery, RVA right vertebral artery, BA basilar artery, (+) migraine with aura, (-) migraine without aura.

risk of stroke (RR 2.76), especially in oral contraceptive users (RR 8.72), patients with visual aura, and cigarette smokers<sup>(5)</sup>.

Even though we know migraines may be related to ischemic stroke, the exact mechanism is still unproven. Two different hypotheses of migraine pathophysiology are well known, one being the vascular theory proposed by Harold Wolff which has fallen out of favor and is replaced by the neurovascular theory. Cortical spreading depression (CSD) is thought to be the trigger of aura in migraine. The release of potassium or glutamate from neural tissue will depolarize the adjacent tissue, which will release more neurotransmitters, resulting in propagating the spreading depression. Two recent studies by Piilgaard and Lauritzen and Chang et al. show that a distinct phase of long lasting mismatch between vascular supply and demand is present in the CSD wave. This will lead to tissue hypoxia and hemoglobin desaturation. The trigeminal system is then activated with the release of calcitonin gene-related peptide (CGRP), a potential mediator of headache pain.

Migraine is independent risk factor for stroke. The evidence was supported in the CAMERA study disclosing a 7-fold increase in the prevalence of silent infarct-like lesions at cerebellum in migraine patients. This phenomenon was more significant in patients with aura and high frequency of attacks<sup>(6)</sup>. The relationship between

women having migraine with aura and late-life prevalence of cerebellar infarct-like lesion was significant, with an odds ratio of almost 29. To the best of our knowledge, the mechanism linking migraines with aura and cerebellar lesions is still a matter of speculation.

The pathogenesis of basilar-type migraine and its correlation with artery dissection remains unclear. Migraine may be related to both intra- and extra-cranial artery dissections<sup>(9,10)</sup>, possibly because edema of the vessel wall during a migraine attack makes the involved artery more vulnerable to tearing. In most cases, however, dissections are related to sudden or unusual stretching of arteries, both in the neck and in the head, regardless of whether there is an underlying abnormality in the connective tissue of the arteries<sup>(3)</sup>. We reviewed the available literature and found ten case reports of migraine related cervical artery dissection involving both carotid and vertebra-basilar arteries and two case control studies supported that an underlying arterial wall injury from repeated migraine attacks could be a predisposing condition for non-traumatic cervical artery dissection. (Table 1). Further studies focusing on the pathogenesis of migraine are necessary for better understanding of its effect on arterial wall.

Artery dissection treatment is controversial. Early anticoagulation resulted in better prognosis; therefore, early diagnosis is important. Because of the side effects

of anticoagulants, especially bleeding, some patients prefer anti-platelet medication. A comparison in clinical outcome between the use of anti-platelet and anticoagulants has not yet been established<sup>(11)</sup>. The patient's clinical symptoms and signs improved with anti-platelet treatment, and the computer tomography angiography follow-up 6 weeks later revealed a complete recanalization of the dissected vessels. This supported the diagnosis of basilar-type migraine and vertebral artery dissection.

We had selected topiramate as prophylactic medication for the migraine and the patient reported no relapse of headache. This suggested that topiramate may be beneficial in prophylactic treatment. Topiramate is an effective neuroprotective agent that may extend the therapeutic window of hypoxic-ischemic rats with delayed hypothermia<sup>(12)</sup>. Besides, one animal study showed that topiramate was able to inhibit regional blood flow changes and cortical depolarization and spreading depression<sup>(13)</sup>. Furthermore, topiramate may successfully reduce attack frequency, and has been shown to be effective for migraine prophylaxis in several multicentre placebo controlled trials<sup>(14-16)</sup>. If repeated migraine attacks may lead to the edematous changes of the involved vessels and cause vessels dissection, reducing the frequency of migraine attacks may lower the possibility of cervical vessel dissection; however, more evidence is needed to support this hypothesis.

In conclusion, even though the exact mechanism has yet to be proven, we propose that migraine-related artery dissection may be caused by vessel wall edematous changes with repeated migraine attacks resulting in sudden or unusual stretching.

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