

## CASE REPORTS

# Limb-shaking transient ischemic attack responsive to nimodipine: A case report

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### Abstract

Limb-shaking transient ischemic attack (TIA), a rare manifestation, is commonly caused by severe stenosis or occlusion of an extracranial internal carotid artery. Such patients are usually treated with surgical revascularization or anti-platelet therapy. We present a 56-year-old woman with 6 months' episodic attacks starting with mouth skewed to the right and a sensation of 'weakness' involving predominantly her left arm, and at times, also involved the left leg. This was immediately followed by rhythmic jerky movements of the left arm and at times, also involved the left leg. Magnetic resonance angiography revealed severe stenosis of M1 segment of the right middle cerebral artery. The patient's symptoms were significantly improved by treatment with anti-platelet drugs and nimodipine.

### INTRODUCTION

Limb shaking transient ischemic attack (TIA) is a rare form of TIA that presents as brief, jerky, coarse, involuntary movements of an arm or leg or both<sup>1</sup>, which always pose a diagnostic confusion with focal motor seizures.<sup>2-4</sup> Up to date, almost all reported limb-shaking TIAs have been associated with a severe stenosis or occlusion of the internal carotid artery (ICA) except occasional cases with anterior cerebral artery (ACA) stenosis<sup>5</sup>, middle cerebral artery (MCA) stenosis together with ACA stenosis<sup>6</sup>, or moyamoya disease.<sup>7</sup> This TIA is relieved by surgical revascularization in some cases<sup>1,3,8</sup> and anti-platelet therapy in others.<sup>4,6,9</sup> We present here a patient with limb-shaking TIA caused by MCA stenosis, with good response to nimodipine, a calcium channel antagonist.

### CASE REPORT

A 56-year-old woman presented with an episodic shaking movements of the left arm for 6 months. These recurrent episodes would usually begin with mouth skewed to the right and a sensation of 'weakness' involving predominantly her left arm, and at times, also involving the left leg. This was followed by the involuntary arrhythmic shaking movements of her left arm and at times, also involving the left leg. There was no involvement

of other body parts. Each episode would last for about 1-3 minutes with duration increased to 5-10 minutes in the month before presentation. Her consciousness was never impaired and there was no other symptom. These attacks would occur while she was standing or walking but never while she was recumbent. There were 1-2 attacks per day with increased frequency to 3-4 attacks per day in the month preceding presentation. She had no past history of diabetics or hypertension.

On examination, she was alert, oriented and anxious, with blood pressure (BP) of 130/80 mmHg, regular heart beat of 68 beats/min, and temperature of 36.8°C. Her neurological and general examination was normal. There was no orthostatic hypotension. The ambulatory electroencephalographic (AEEG) monitoring was normal. MRI including diffusion-weighted MRI of the brain was normal. Magnetic resonance angiography (MRA) showed a focal stenosis of the M1 segment of the right middle cerebral artery (MCA) (Figure 1A). Extracranial segment of the right internal carotid artery (ICA) was normal. Ultrasound studies did not reveal any micro-embolic signal in the right MCA and ICA while the patient was asymptomatic. The recurrent event of this patient was diagnosed as TIA<sup>10</sup> secondary to right MCA stenosis.

The patient refused further cerebral angiography for possible surgical revascularization or stenting. She was treated with acetylsalicylic acid (300 mg once daily) and clopidogrel (75 mg once daily) and advised to avoid prolonged standing and walking. After commencing the anti-platelet medications, the recurrent attacks persisted with the attack frequency unchanged but the duration reduced to 1-3 minutes. Based on the consideration that its effect on smooth muscle may relax the cerebrovasculature and improve blood flow, nimodipine was added at a dose of 30 mg every 6 hours, two weeks after commencing the anti-platelet medications. The attack frequency reduced to 3 attacks in a week. The nimodipine dose was increased to 60 mg every 6 hours, and she reported no attack for one week. A follow up MRA two weeks after commencing nimodipine, one week after the dose was increased, showed a significant improvement of MCA stenosis and the blood supply in MCA territory (Figure 1B). During the following 4 months, only 1-2 attacks per month were reported with the attack duration of 1-3 minutes.

## DISCUSSION

An episodic limb shaking syndrome associated with carotid stenosis was first described by Miller Fisher in 1962.<sup>11</sup> Since then, limb-shaking TIA

has been reported regularly and to date 55 cases were published in case reports<sup>10</sup> and 34 cases in case control.<sup>12</sup> Limb-shaking TIA usually consists of rhythmic or arrhythmic involuntary hyperkinesias, which patients describe as shaking, jerking, or twitching, affecting the unilateral hand, arm, leg, hand-arm, or hand-arm-leg with face spared. In our case, the the attack was described as starting with the face pulled to the right. This was misdiagnosed as focal seizures in another medical center. She was also given carbamazepine without improvement of symptoms. The associated weakness of the left arms suggested that the patient may have TIA. This was supported by the MRA findings of right MCA stenosis.

In our patient, the attacks occurred exclusively during standing or in walking and never when she was recumbent. The attacks ceased when she sat or lie down, indicating the role of cerebral hypoperfusion in causing the symptom. Cerebral blood hypoperfusion from ICA stenosis has long time been assumed to be the underlying pathophysiological mechanism of the limb-shaking TIA. The hypoperfusion theory is confirmed by a recent case control study where the patients who have ICA occlusion and limb-shaking attacks were shown to have impaired cerebral blood flow affecting the border zone between ACA and MCA territory, as compared to those patients who have only ICA occlusion

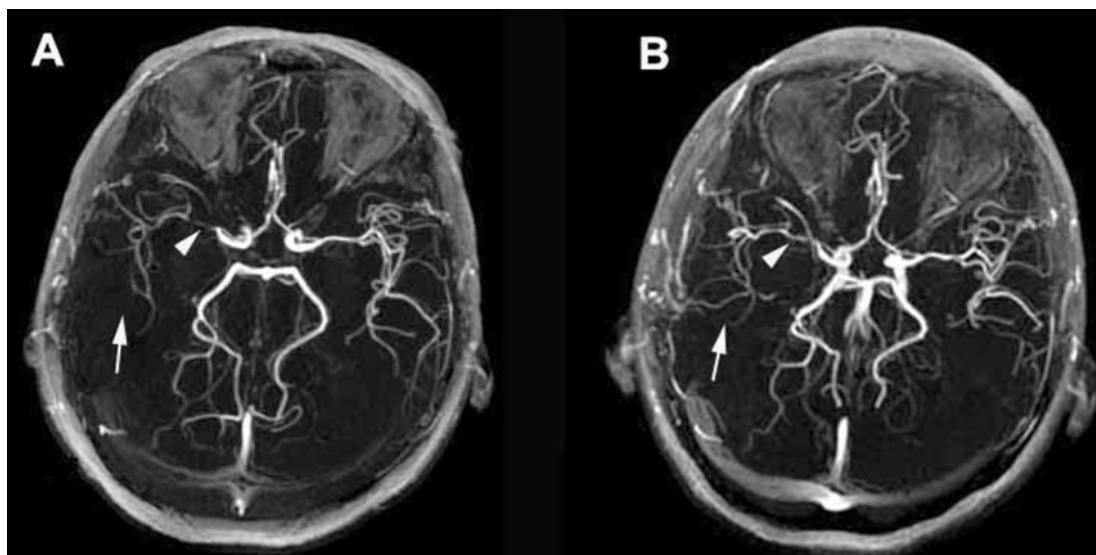


Figure 1. A. Magnetic resonance angiogram (MRA) shows severe focal stenosis (arrowhead) of the M1 segment of the right middle cerebral artery, with fewer blood vessels distal to the stenosis (arrow) when compared to the left side. Both anterior cerebral arteries and posterior cerebral arteries are normal. B. Follow-up MRA two weeks after initiation of nimodipine shows improvement in the stenosis of the M1 segment of the right middle cerebral artery (arrowhead), with more blood vessels seen distal to the previous stenosis (arrow).

but no limb-shaking attacks.<sup>12</sup> A significant MCA stenosis but not ICA occlusion or stenosis was found in our patient. The clinical symptom significantly improved after treatment, which was associated with improvement in the corresponding cerebral perfusion. There is only one previous report of limb-shaking TIA with MCA stenosis and absence of both ACA, where hypoperfusion in the border zone between the ACA and the MCA territory was detected by perfusion CT.<sup>6</sup> We believe that the limb-shaking TIA associated with MCA stenosis in our patient may also share the similar mechanism with other limb-shaking TIA associated with ICA occlusion or stenosis.

Management of limb-shaking TIA focuses on maintaining or improving cerebral blood flow by careful elevation of blood pressure and surgical revascularization.<sup>4</sup> Because our patient refused surgical revascularization, she was given anti-platelet drugs 300 mg of acetylsalicylic acid and 75 mg of clopidogrel once daily. This did not improve the clinical symptom, though it has been shown to be effective in previous case reports.<sup>4,6</sup> Our patient showed dramatic improvement with the supplement of calcium channel blocker, nimodipine, reducing the frequency and intensity of the limb-shaking attacks. Our patient's response to nimodipine was impressive as it occurred when the TIA were still worsening. The close temporal relationship between the relief of TIA attacks and initiation of oral nimodipine was in favor of the direct therapeutic effect of nimodipine. Nimodipine is currently recommended for vasospasm-associated disorders based on its ability to relax the smooth muscle of arterial walls.<sup>13,14</sup> The dramatic improvement of MCA stenosis on MRA after nimodipine treatment may also implicate vasospasm, with or superimposed on atherosclerosis, as a mechanism of stenosis in this patient. This is because the improvement is unlikely to be due to a reversal of atherosclerosis in the short period of two weeks. The recurrent TIA in our patient may at least partially, be due to vasospasm in the MCA territory, though the atherosclerosis is usually a common cause of cerebral artery stenosis. This is supported by reports of cerebral vasospasm in association with atherosclerosis.<sup>15,16</sup>

To our knowledge this is the first case report of limb-shaking TIA caused by MCA stenosis responsive to nimodipine. This patient shows that anti-platelet medications combined with nimodipine therapy may be a therapeutic option for patients with limb-shaking TIA caused by MCA stenosis.

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## DISCLOSURE

Conflict of interest: None

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