# A young lady with thalamic stroke mimicking acute Miller Fisher syndrome

***Shariful Hasan,*** *MBBS, FCNp,*
***Johnson Stanslas,*** *MSc, PhD,*
***Lim P. Hin,*** *MD, ABPN (Dip),*
***Hamidon B. Basri,*** *MD, MMed.*

Diagnosis of acute ischemic stroke is usually straightforward, though this may not always be the case. Sometimes non-vascular conditions may present with a clinical picture that simulates stroke (stroke mimics), while at other times stroke may mimic non-vascular conditions. Hypoglycemia, hyperglycemia, epilepsy, multiple sclerosis, hemiplegic migraine, intracranial tumor and infections are the most common and important causes of stroke mimics. Huff[1] coined the term 'stroke chameleon' for strokes that take on the appearance of some other conditions. In clinical practice stroke chameleons are less common than stroke mimics.[1] Unusual presentation of stroke is difficult to diagnose, especially if it is in a young patient with no predisposing risk factor. The objective of this report is to highlight a young female patient with stroke chameleon, which mimicked the presentation of acute Miller Fisher syndrome.

A 23-year-old lady presented with complaints of giddiness, difficulty in looking upwards, and unsteady gait. She denied any history of abnormal sensation or altered consciousness. There was neither any history of risk factors for vascular events, nor any significant past medical history other than common cold 3 weeks prior to current illness. She also did not have any significant family history related to this illness. On examination, her vital signs were normal. She was alert and had no evidence of language disorder. Cranial nerve examination revealed complete vertical gaze palsy and bilateral first-degree nystagmus. Clinical examination of the motor system and gait showed reduced reflexes, bilateral flexor plantar response, and mild difficulty in tandem walking. There were no abnormalities in muscle tone, power, pupil, and vision. Besides mild difficulty in tandem walking, there were no other cerebellar signs. An MRI of the brain carried out on the same day did not show any abnormality. Investigations including full blood count, renal profile, liver function test, fasting blood glucose, lipid profile, anti-nuclear antibodies, Venereal Disease Research Laboratory test (VDRL), protein C and S levels, echocardiography, Holter monitoring, and nerve conduction study (NCS) were carried out. There was no significant finding in any of the investigations. She was diagnosed as possible Miller Fisher syndrome considering the age of the patient, clinical history, normal MRI, and the absence of risk factors for vascular events. She was managed conservatively. However, MRI of the orbit (and brainstem) carried out 3 days later as requested by an Ophthalmologist revealed a small hypo-intense lesion in apparent diffusion coefficient (ADC) and hyper-intense lesion in T2 weighted imaging (T2WI) in the right medial thalamus (**Figure 1**). This finding changed our initial diagnosis to right thalamic ischemic stroke. The standard treatment for ischemic stroke of aspirin 150 mg/day was given. She recovered completely in one month. On subsequent follow ups, she was found stable, healthy, and without any complication of previous stroke.

Miller Fisher syndrome is an autoimmune disorder, which is commonly preceded by a viral infection. It is an acute polyneuropathy that presents with a clinical triad of ophthalmoplegia, ataxia, and areflexia. It may sometimes present with 2, or even a single feature. Although our patient had hyporeflexia and not areflexia, she fulfilled the other 2 criteria of the triad of Miller Fisher syndrome. Anti-ganglioside antibody (Anti-GQ1b) is present in 85% cases of Miller Fisher syndrome, though it is not strictly specific. Moreover, in countries like Malaysia, where Anti-GQ1b test is not readily available, the test is carried out abroad, which requires additional time and cost. It is not impossible to have normal NCS finding in Miller Fisher syndrome if carried out very early, which may explain the normal NCS finding in our patient. It was also too early to do CSF analysis in this patient. Furthermore, a normal MRI was reassuring for a lack of other possible pathology. Therefore, the diagnosis of Miller Fisher syndrome was

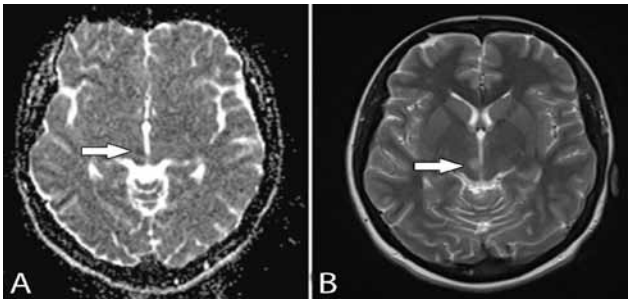


**Figure 1 -** An MRI of the brain showing: **A**) hypo-intense lesion in apparent diffusion coefficient; and **B**) hyper-intense lesion in T2 weighted imaging at the site of right medial thalamus.

**Disclosure.** The authors declare no conflicting interests, support or funding from any agency in the public, commercial, or not-for-profit sectors.

made initially. However, an MRI scan repeated after 3 days that demonstrated a thalamic infarct led us to change our initial diagnosis. Nevertheless, questions can be raised, whether this is a subset of Anti-GQ1b syndrome, which may involve brainstem, thalamus, cerebellum, or white matter of the cerebrum. These syndromes usually affect the level of consciousness of the patient, which was absent in our patient. Cases of stroke where initial imaging failed to show any abnormality, but subsequent imaging demonstrated the lesions are not uncommon. The classical presentation of thalamic infarct is hemisensory loss. The presentation in our case was not very typical of a thalamic infarct. The clinical picture of thalamic infarct depends on the thalamic nuclei involved in the vascular lesion. The vertical gaze impairment seen in our patient is more often seen with midbrain lesions, or lesions of the thalamus that extend to the midbrain rather than lesions of the thalamus per se. It is usually associated with the lesions of the mesencephalic reticular formation including the nucleus of Darkschewitsch, interstitial nucleus of medial longitudinal fasciculus, interstitial nucleus of Cajal and posterior commissure. When vertical gaze palsies occur in thalamic lesions, they are usually attributed to coexisting lesions of the rostral midbrain. However, it is possible that some of the clinical phenomena may result from a lesion of the fiber pathways rather than lesions of the nuclei themselves. For instance, a lesion of the medial thalamus that causes disruption of the corticofugal fibers, which lead from motor and premotor cortices to the nucleus of Darkschewitsch and interstitial nucleus of Cajal in the midbrain, may be responsible for vertical gaze palsy.[2] This is further supported by Lazzaro et al,[3] who described thalamic infarctions without midbrain involvement. Vertical gaze palsy without midbrain involvement may be due to either the midbrain lesions being too small to be detected by imaging, or it could be the thalamic lesion that caused the vertical gaze palsy by interrupting supranuclear inputs.

Carrera et al[4] confirmed that isolated paramedian or anteromedian thalamic infarcts can interrupt supranuclear tracts responsible for vertical gaze control without involvement of mesencephalon. The mechanism of vertical gaze palsy with unilateral lesions in thalamus is not very clear. However, it is possible because of decussation of the frontobulbar fibers in the medial thalamus. In a more recent study by Hermann et al,[5] vertical gaze palsy was one of the presenting features in 76% of the paramedian thalamic infarct patients. In the same study, 67% of the patients had mild gait ataxia, which was also a presenting feature in our patient. Lesions of the thalamus are thought to be associated with better prognosis than those of the cerebral cortex or other cortical structures. Hermann et al[5] also demonstrated that stroke outcome is usually better in right-sided thalamic lesions than in bilateral or left sided infarcts. In our case, the patient had a right-sided lesion, and she recovered completely. Although there was a little delay in diagnosis, it did not affect the final outcome.

This case highlights an unusual presentation of thalamic infarct in a young patient without any vascular risk factors. It should be noted that presentation may vary, depending on which nuclei of the thalamus are affected. It is imperative to realize that normal imaging does not always exclude a vascular etiology, especially if performed very early. A repeat brain MRI with special attention on the regions corresponding to the clinical signs should be encouraged in these cases.

**Acknowledgments.** *We extend our thanks to MakroCare, for the editing services. Likewise to Dr. Sumithra Ranganathan, Consultant Radiologist, Prince Court Medical Centre, Kuala Lumpur, Malaysia for reporting the brain MRI, and Dr. Rafid Salim Jabir, Pharmacotherapeutics Unit, Faculty of Medicine and Health Sciences, University Putra Malaysia for the Arabic translation.*

*Received 28th February 2012. Accepted 24th June 2012.*

*From the Neurology Unit (Hasan, Hin, Basri), Department of Medicine, Institute of Gerontology (Hasan), and Pharmacotherapeutics Unit (Stanslas), Faculty of Medicine and Health Sciences, University Putra Malaysia, Selangor Darul Ehsan, Malaysia. Address correspondence and reprint requests to: Dr. Hamidon B. Basri, Deputy Dean and Head of Neurology Unit, Department of Medicine, Faculty of Medicine and Health Sciences, University Putra Malaysia, Serdang 43400, Selangor Darul Ehsan, Malaysia. Tel. +603 (8) 9472607. Fax. +603 (8) 9472759. E-mail: shariful_hsn@yahoo.com*

## References

1. Huff JS. Stroke mimics and chameleons. ***Emerg Med Clin North Am*** 2002; 20: 583-595.
2. Schmahmann JD. Vascular syndromes of the thalamus. ***Stroke*** 2003; 34: 2264-2278.
3. Lazzaro NA, Wright B, Castillo M, Fischbein NJ, Glastonbury CM, Hildenbrand PG, et al. Artery of percheron infarction: imaging patterns and clinical spectrum. ***AJNR Am J Neuroradiol*** 2010; 31: 1283-1289.
4. Carrera E, Michel P, Bogousslavsky J. Anteromedian, central, and posterolateral infarcts of the thalamus: three variant types. ***Stroke*** 2004; 35: 2826-2831.
5. Hermann DM, Siccoli M, Brugger P, Wachter K, Mathis J, Achermann P, et al. Evolution of neurological, neuropsychological and sleep-wake disturbances after paramedian thalamic stroke. ***Stroke*** 2008; 39: 62-68.