

Case Report

Bilateral Paramedian Thalamic Infarcts after Occlusion of the Artery of Percheron

Shibchurn Mithilesh*

Department of Neurology, Zhejiang University, China

Abstract

Study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript and critical revision of the manuscript for important intellectual content.

Keywords: Diabetes mellitus; Percheron; Hyperlipidemia

Introduction

A 76-year-old woman was admitted after being found to be comatose in bed with a Glasgow Coma Scale of 8. She has a long history of diabetes mellitus, hyperlipidemia, and hypertension and has also been on regular medication to control her atrial fibrillation. Emergently performed Magnetic Resonance Imaging-MRI showed symmetric bilateral hyperintense thalamic lesions (Figure 1A). Anticoagulation was promptly started, and she was sent to the Intensive Care Unit-ICU. She regained consciousness after two days. However, she exhibited fluctuating level of consciousness. Neurological deficits like paresis, confusion, disorientation, dysarthria, hypophonia and memory impairment were present. Lower extremities doppler ultrasound showed poor blood flow in the left posterior tibial veins indicating Deep Venous Thrombosis; DVT (Figure 1B). Additionally, bedside echocardiogram demonstrated a hypochoic mass of 28.6 mm × 20.8 mm in the left atrium Figure 2 (thrombosis cannot be excluded). Unfortunately, due to personal reasons her family members did not wish to continue treatment and we could not do further follow up.

Discussion

The thalamus is part of the brain which is found in the posterior region of the forebrain more precisely in the diencephalon. There are three possible ways for the posterior blood supply to feed the thalamus (Figure 3A) [1]. Bilateral thalamic infarcts are uncommon types of ischemic strokes and are rare in medical literature but reports a range of 0.1% to 2% in all ischemic strokes and 4% to 18% in thalamic infarction. Because our patient had a bilateral thalamic infarction, I suspect that this thalamic vascular supply was a type 2 variation. In type 2, the thalami are supplied by the Artery of Percheron (AOP). The latter is further divided into four major thalamic vascular territories; Tuberothalamic artery, Paramedian artery, Thalamogeniculate artery

and Posterior choroidal artery (Figure 3B). Vascular lesions of the AOP causes bilateral thalamic infarction and destroys the thalamic nuclei which produces sensorimotor and behavioral syndromes which depends on the affected nuclei. The thalamic nuclei are responsible for five major functions. Reticular and intralaminar nuclei are vital for arousal and nociception; sensory nuclei are essential for all major domains; effector nuclei are significant for motor functions; associative nuclei are responsible for high-level cognitive functions and aspects of language; limbic nuclei are important for normal functioning of mood and motivation. Loss of these functional classes leads to neuropsychological manifestations such as disorientation, confusion, hypersomnolence, deep coma, memory impairment and apathy. Subarachnoid Hemorrhage (SAH) should not be confused with occlusion of AOP because it causes sudden onset of lethargy, paresis, decreasing level of consciousness, comatose state in the absence of motor deficits which easily evokes the idea of SAH [1]. Besides, odor abnormalities are often overlooked because this is usually hindered by mental disturbances in such patients [2]. According to medical analysis 60% of thalamic infarctions arise from small artery disease and 40% is followed by cardioembolism [1].

In conclusion, AOP occlusion is a rare cause of coma and should be suspected in patients who present with acute loss of consciousness only if MRI shows acute infarction in symmetrical bilateral thalami which may be due to unpaired thalamoperforating artery trunk arising from the first part of the posterior cerebral artery and supplies bilateral medial thalami. Moreover, we believe that such cases have been overlooked without the widespread use of MRI. AOP should be considered in patients presenting with symmetrical infarction of both thalami.

References

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***Corresponding author:** Shibchurn Mithilesh, Department of Neurology, Sir Run Run Shaw Hospital, College of Medicine, Zhejiang University, China, E-mail: mithileshshibchurn@yahoo.com

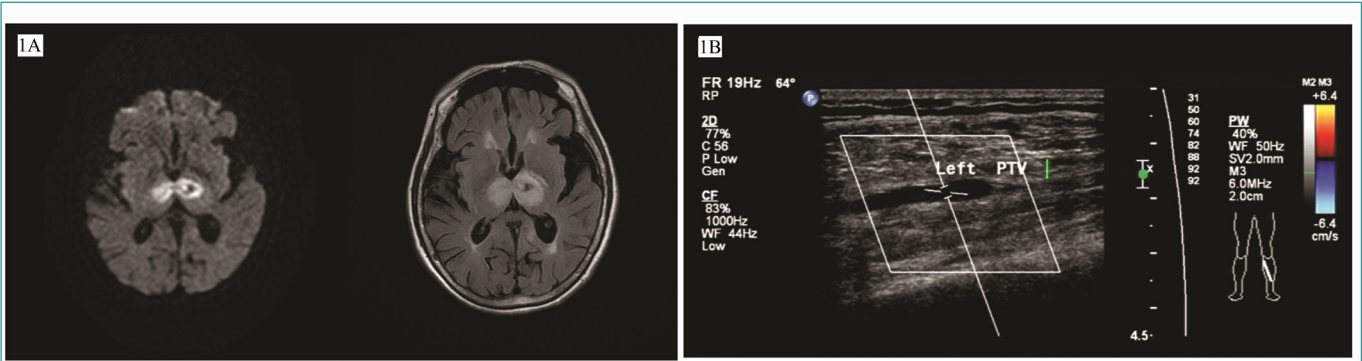


Figure 1: A) Axial diffusion-weighted MR image of brain & Axial T2-weighted MR FLAIR image showing bilateral hyperintensity in the thalami indicating of infarction. B) Intraluminal echogenicity in the left posterior tibial vein showing DVT.

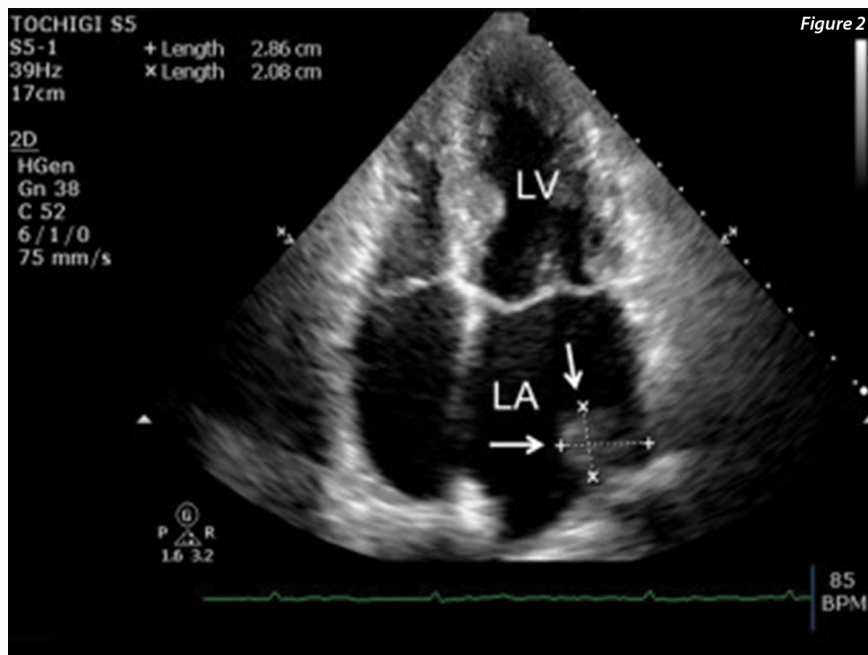


Figure 2: Echocardiogram showing hypoechoic mass in the left atrium.

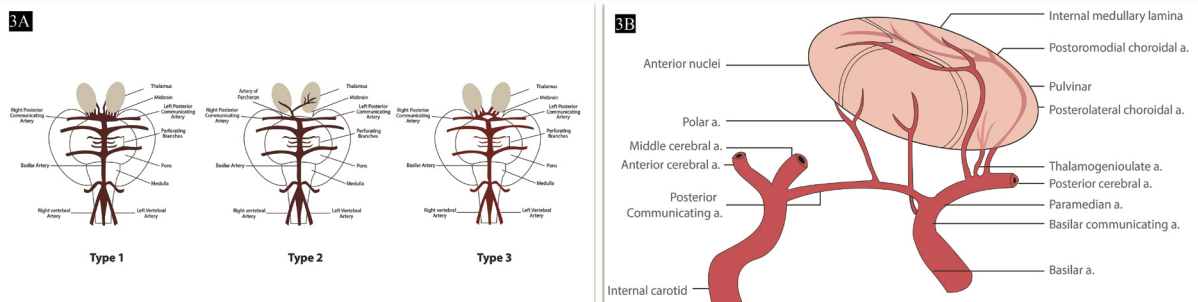


Figure 3: A) Posterior blood supply to feed the thalamus. B) Thalamogeniculate artery and posterior choroal artery.