Bilateral Symmetrical Thalamic Lesions: An Infarction Involving the Artery of Percheron

Ewe Jin Koh[1]*, Alvin John Gnanasegaran[2] and Ming Lee Chin[3]

[1]Internal Medicine Specialist, Taiping Hospital, Perak, Malaysia

[2]Medical Officer, Taiping Hospital, Perak, Malaysia

[3]Specialist, Taiping Hospital, Perak, Malaysia

Received: 23 May 2023

Accepted: 13 June 2023

*Corresponding author: ewejinkoh@gmail.com

DOI 10.5001/omj.2025.03

Abstract

The artery of Percheron is a rare anatomical variant artery that supplies the bilateral thalami and midbrain. An acute occlusion of the vessel will lead to the presentation of a bilateral thalamic stroke syndrome. The classical presentation includes somnolence and reduced level of consciousness. Therefore this clinical entity often goes unrecognized and diagnoses often delayed due to the ambiguity of presenting symptoms. We report a case of a gentleman presenting with acute onset drowsiness, later to be diagnosed to have a bilateral thalamic infarct only days later due to the vague clinical symptoms. The authors also describe several learning points of this case and the importance of recognizing this syndrome.

Introduction

In 1973, Gerald Percheron, a French medical scientist examined 50 cadaveric brain specimens in attempts to further elucidate the anatomy and the arterial supply to the thalamus. At that time, the thalamus was recognized as an important structure for everyday function, but was still lacking clarity. He had described a variant artery, branching off from the basilar artery to the junction of the posterior communicating artery and had proposed to call it the 'communicating basilar artery'[1]. This artery was later eponymously named after him, and was termed the 'artery of Percheron'. The clinical significance lies in the fact that an occlusion of this artery will lead to the phenomenon of a bilateral thalamic stroke. However, recognition of this pathology often goes unnoticed because of the vague clinical presentation, often without any focal neurological signs, potentially delaying timely thrombolytic treatment.

Case Report

A 68 year old gentleman with Type 2 diabetes mellitus presented to the emergency department with acute onset of reduced consciousness witnessed by his wife when he was not arousable after taking his routine afternoon nap. He had otherwise been fully conscious the morning of his presentation. He was premorbidly a high functioning gentleman who was still completely independent in his activities of daily living.

There was no preceding fever or constitutional symptoms. The family had denied any recent travel and to the best of their knowledge were unaware of any substance or illicit drug use.

On assessment, he was predominantly drowsy, scoring nine out of fifteen on the Glasgow Coma scale (GCS). Vital signs were otherwise unremarkable. He was breathing comfortably under ambient air. Pupils were equal bilaterally and there was no obvious focal localizing neurological signs. His blood work was unremarkable as were his blood sugars. His electrocardiogram and urine toxicology screen were normal. A plain computed

tomography (CT) of the brain was normal, which could not explain the reason for the acute onset reduced drowsiness (Figure 1).

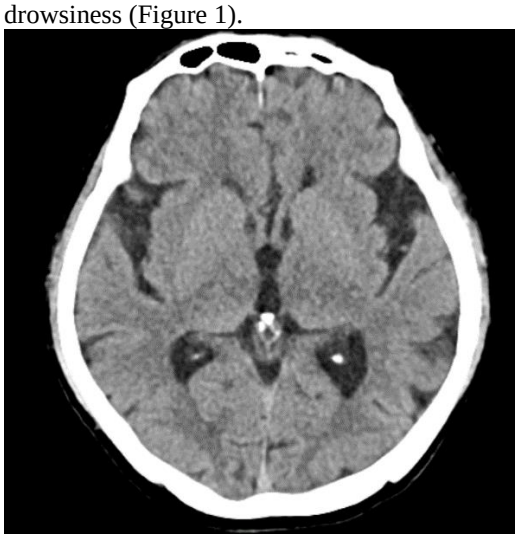

Figure 1: Plain CT Brain showing no evidence of any acute infarction.

The gentleman was admitted to the ward with the impression of acute encephalopathy with no identifiable cause. A lumbar puncture was eventually performed on the first day of admission. Cerebrospinal fluid (CSF) protein was only marginally elevated at 0.54g/L. The CSF glucose, cultures and cytology were unremarkable.

As there was no identifiable cause or diagnoses from these preliminary investigations, a magnetic resonance imaging (MRI) of the brain was performed within 48 hours of his presentation. This revealed revealed symmetrical hyperintense lesions at bilateral thalami on axial T2-FLAIR and on diffusion weighed imaging (DWI) with corresponding hypointensities on apparent diffusion coefficient (ADC) suggestive of an acute bilateral thalamic stroke (Figure 2).

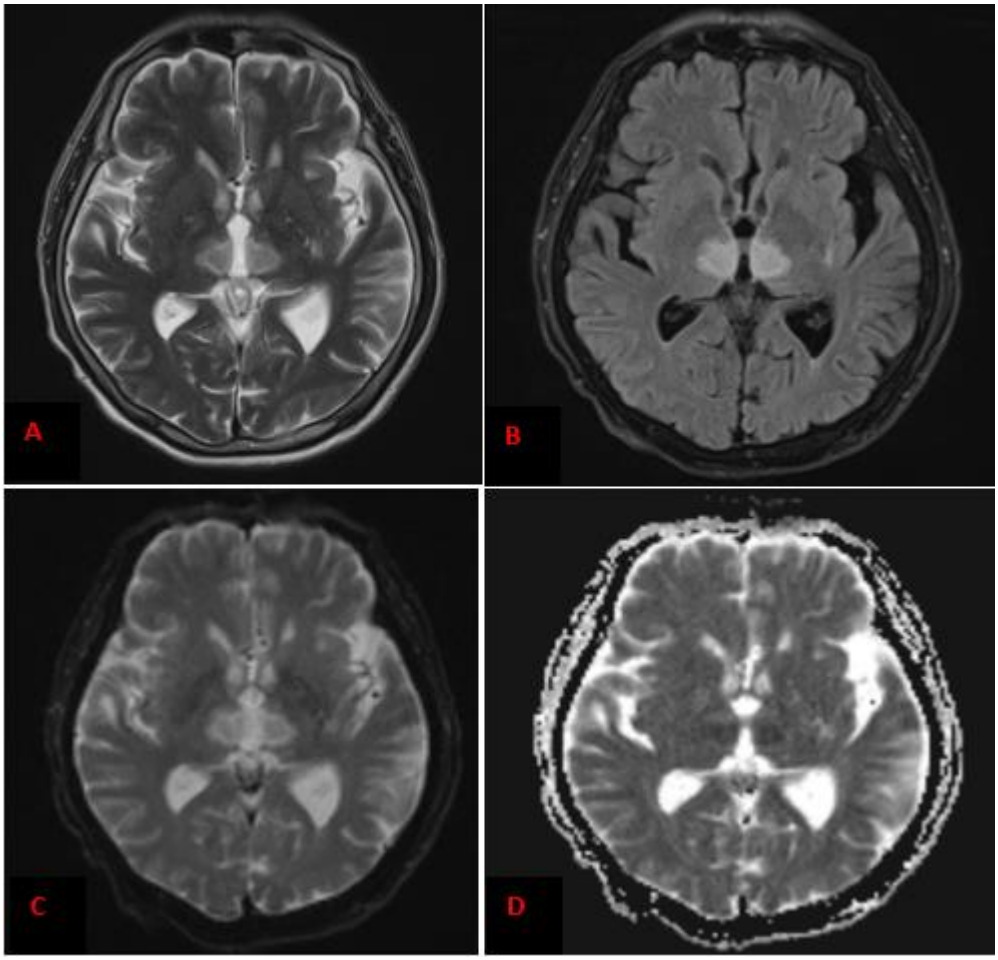

Figure 2: MRI Brain images. T2WI, axial FLAIR and DWI showing hyperintensies at bilateral thalami (A,B and C). ADC showing hypointensities at bilateral thalami.

Correlating his acute presentation with corresponding findings on the MRI brain, the patient was diagnosed to have an acute bilateral thalamic stroke. An infarction involving the artery of Percheron was suspected due to the symmetrical distribution of the infarct over the bilateral thalami. However, we were unable to perform an angiogram due to cost and financial circumstances.

The patient was started on medications for secondary stroke prevention with antiplatelets and also a statin. He was eventually discharged with home care and support with fluctuations in his consciousness after a period of rehabilitation and carer training.

Discussion

The artery of Percheron is a rare anatomical variant in which the vessel arises from the posterior cerebral artery and provides the only blood supply to the bilateral paramedian thalami and rostral midbrain[2]. This was first described by Gerald Percheron, a French scientist who studied thalamic blood supply extensively in the twentieth century. In normal anatomy, the P1 segment of the posterior cerebral artery (PCA) supplies the paramedian thalamus through the paramedian arteries on each side. Paramedian arteries may sometimes arise from one common trunk from a P1 segment on either side: this common trunk is eponymously known as the artery of Percheron. Its prevalence is estimated to be occur in about 7-11% of the general population[3]. A bilateral paramedian thalamic infarct with or without the involvement of the rostral midbrain can result if the blood supply to the AOP is compromised[4]. Thrombosis of this vessel constitutes only 0.1-2.0% of all acute ischemic stroke presentations. Classical symptoms include acute impairment in arousal with vertical gaze palsies. There are usually no accompanying focal neurological and pyramidal signs unlike classical stroke syndromes[5]. Initial plain CT scans of the brain may be normal. Therefore, due to the ambiguity of symptoms, diagnosis may be challenging especially in the acute setting. This is illustrated in the following figure below (Figure 3)[6].

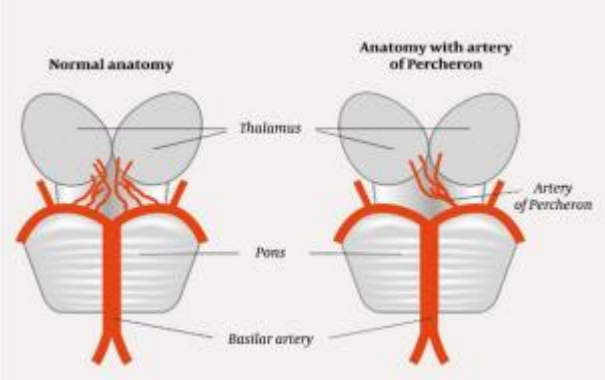

Figure 3: Schematic diagram illustrating the normal anatomical arterial supply to the bilateral thalami (Left) vs the variant anatomy (Right).

There are a vast differential diagnoses for bilateral symmetrical thalamic lesions. These include vascular disorders such as arterial ischemia and venous infarctions, but also different tumors, infectious and demyelinating diseases, as well as metabolic-toxic alterations[7]. A good clinical history encompassing the acuteness of the onset of the presentation, correlated with radiological findings are important in narrowing down the differential diagnosis. More often than not, diagnosis is often delayed and patients are routinely subjected to many invasive and investigative procedures which are justified in order to rule out other possible differential diagnoses.

Conclusion

This case serves to highlight that a high clinical suspicion is needed and recognition of this rare entity is necessary to avoid unnecessary investigations and treatment decisions that skew the clinicians' decisions away from that of an acute stroke. Patients who present within the therapeutic time frame may also benefit from thrombolytic therapy if this clinical syndrome is promptly recognized and diagnosed.

Acknowledgements

The authors would like to thank Dr Chee Yong Chuan, Neurologist from Hospital Universiti Sains Malaysia (HUSM) for guidance on interpreting the MRI images.

References

1. Percheron G. The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. Z Neurol [Internet]. 1973;205(1):1–13. Available from: http://dx.doi.org/10.1007/bf00315956

2. Ranasinghe KMIU, Herath HMMTB, Dissanayake D, Seneviratne M. Artery of Percheron infarction presenting as nuclear third nerve palsy and transient loss of consciousness: a case report. BMC Neurol [Internet]. 2020;20(1):320. Available from: http://dx.doi.org/10.1186/s12883-020-01889-

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 [Internet]. 2010;31(7):1283–9. Available from: http://dx.doi.org/10.3174/ajnr.A2044

4. Morais J, Oliveira AA, Burmester I, Pires O. Artery of Percheron infarct: a diagnostic challenge. BMJ Case Rep [Internet]. 2021;14(4). Available from: http://dx.doi.org/10.1136/bcr-2020-236189

5.Jiménez Caballero PE. Bilateral paramedian thalamic artery infarcts: Report of 10 cases. J Stroke Cerebrovasc Dis [Internet]. 2010;19(4):283–9. Available from: http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2009.07.003

6. Nadeeshani PGN, Liyanage A, Rajendiran T, Indrakumar J. Acute bilateral thalamic infarctions with midbrain involvement due to artery of Percheron occlusion. J Ceylon Coll Physicians [Internet]. 2020;51(2):136. Available from: http://dx.doi.org/10.4038/jccp.v51i2.7912

7. Linn J, Danek A, Hoffmann LA, Seelos KC, Brückmann H. Differential diagnosis of bilateral thalamic lesions. Klin Neuroradiol [Internet]. 2007;17(1):3–22. Available from: http://dx.doi.org/10.1007/s00062-007-7000-x