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**Case Report**

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# Artery Of Percheron Infarction: A Great Mimicker

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## ABSTRACT

The thalamus is a deep structure located in the diencephalon, whose arterial blood supply is mainly from four branches of posterior cerebral artery. The artery of percheron (AOP) is an infrequent variation of thalamic perfusion, occlusion of which presents with a heterogenous, atypical list of symptoms without focal signs. This is in contrast to the typical, easily recognizable focal neurology of other ischemic infarcts. Therefore, AOP infarctions may be misdiagnosed, delayed in diagnosis or missed altogether. It is a rare, but vital area of neurology that needs to be studied by clinicians to facilitate an overall care for patients.

We report the case of a 59-year-old lady who presented to us with bilateral complete ptosis with bilateral vertical gaze palsy and nystagmus, whose initial NCCT brain was normal. Subsequent MRI showed bilateral thalamic and midbrain ischemic infarctions in the artery of Percheron distribution. Our case highlights the importance of high degree of suspicion, early diagnosis and role of MRI in the diagnosis when initial CT is normal.

*Keywords: Artery of Percheron, hypersomnolence, ophthalmoplegia, Bithalamic infarction*

## INTRODUCTION

The vascular territories of the thalamus are classified as anterior, paramedian, inferolateral and posterior (1). Blood supply is primarily from the posterior communicating artery (PComA) and posterior cerebral arteries (PCA). Paramedian territories are usually supplied by the posterior thalamosubthalamic branches arising from the interpeduncular segments (P1) of PCAs1. (Figure 1.) Artery of Percheron, first described by Gerard Percheron in 1973, is a rare clinical variant, comprising of a single arterial trunk arising from P1 segment of PCA of one side, supplying paramedian territories of thalami and rostral midbrain bilaterally (2,3). Therefore, lesions of AOP give rise to heterogenous, atypical, bilateral symptoms. The commonly recognized triad being memory impairment, altered mentation and ophthalmoplegia. Patients may also have hemiplegia, cerebellar signs, movement disorders and akinetic mutism (4). This is in contrast to the usual predictable focal neurological signs associated with cortical infarcts. AOP infarction should be considered in a patient with decreased mentation associated with a normal initial CT scan. MRI brain, especially diffusion weighted imaging, is the gold standard for imaging diagnosis of AOP infarct5. Early diagnosis may prevent preventable fatalities and minimize co-morbidities.





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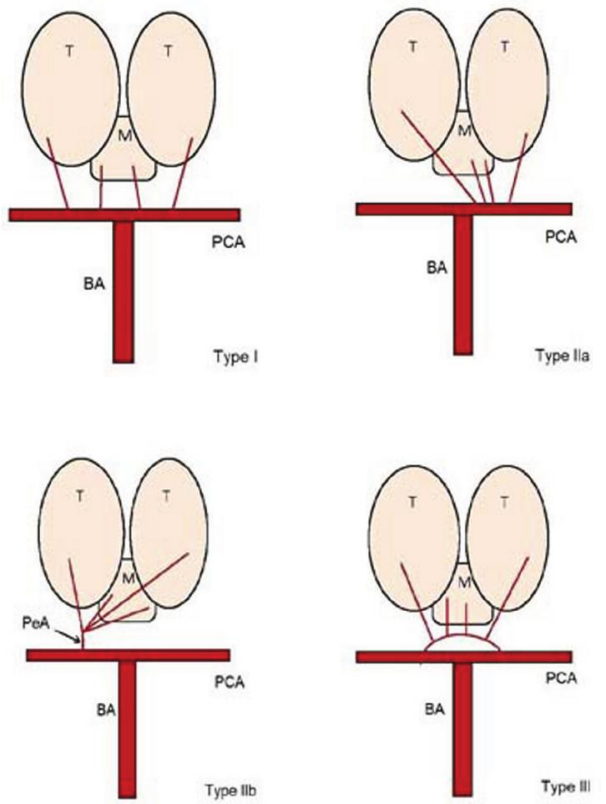


**Figure 1. Variations of perforating paramedian arteries of thalamus**

## CASE REPORT

### Presenting complaint

A 59-year-old lady who was previously diagnosed to be having hypertension and evaluated for chest pain 5 years prior, presented to the medical casualty ward with a history of difficulty opening eyes for 1 day.

### Clinical findings

She developed sudden difficulty opening eyes on the day before admission with associated sleepiness and difficult arousal. Next day, she was found to be unresponsive by family members and brought to the hospital, by which time the responsiveness had significantly improved with patients claiming difficulty in eye opening and memory impairment of the last 3 days. She did not give a history of any chronic/acute headache, seizures, slurred speech, dysphagia, upper or lower limb weakness or numbness, facial asymmetry, bladder or bowel involvement. There was no significant history of fever, similar past neurological defects, features of increased intracranial pressure, history of alcohol use or recurrent vomiting, use of illicit drugs/toxin ingestion, recent hospitalization with fluid resuscitation, or recent change in mentation. Similarly, she did not give a history of snake bites, recent sore throat, viral infection, recent canned food/shellfish ingestion, recent change in medication, history of thyroid or liver disease.

On examination, she was afebrile, not showing signs of meningeal irritation. Neurological examination revealed GCS of 14/15, slight drowsiness, bilateral complete ptosis, with bilateral vertical gaze palsy and bilateral horizontal nystagmus. There was no other cranial nerve involvement, with preservation of bilateral pupillary direct and consensual reflexes and normal fundoscopy. The upper limb and lower limb examination was unremarkable, with preserved sensation and absence of limb cerebellar signs. No evidence of fatigueability or proximal myopathy was found. The ice pack test done at the bedside was negative and gait was preserved. Vital signs were unremarkable. Other system examinations were normal.

### Diagnostic focus and assessment

Urgent investigations planned showed a Capillary blood sugar of 205 mg/dL, and Non-contrast CT brain which did not show any obvious infarctions, hemorrhages or mass lesions. Liver, renal functions and coagulation screen were normal. ECG showed left ventricular hypertrophy (LVH). The rest of the investigations were as follows.

1. Full blood count

Table 01: Investigations of full blood count

| Test | Results | Normal value |
|---|---|---|
| WBC | 6.51 x$10^9$/L | (4-10) x $10^9$/L |
| Neutrophils | 3.7 x $10^9$/L | (2-7) x $10^9$/L |
| Lymphocytes | 2.8 x $10^9$/L | (0.12-2) x $10^9$/L |
| Eosinophils | 0.19 x $0^9$/L | (0.8-4) x $10^9$/L |
| Hemoglobin | 11.4 g/dl | (11-16) g/dl |
| MCV | 88 fL | (76-96) fL |
| Platelets | 187 x $10^9$/L | (150-450) x$10^9$/L |

2. Inflammatory markers

Table 02: Investigations of inflammatory markers

| Test | Results |
|---|---|
| CRP | 2.86 mg/L |
| ESR | 12 mm/1st hour |

3. UFR

Table 03: Investigations of UFR

| Protein | - |
|---|---|
| Sugar | Nil |
| Pus cells | 3-4/HPF |
| Red cells | - |
| Epithelial cells | Few |
| Casts | Hyaline casts |

4. CSF analysis

Table 04: Investigations of CSF analysis

| Appearance | Clear |
|---|---|
| Sugar | 120 mg/dL |
| Protein | 37 mg/dL |
| White blood cells | 4/mm$^3$ with 100% lymphocytes |
| Red cells | 20/mm$^3$ |

5. Nerve conduction test and Repetitive nerve stimulation test- normal.

6. MRI/MRA Brain - Diffusion restriction with high T2 and low T1 signal in bilateral paramedical thalami and changes to a lesser extent in the paramedical midbrain indicative of acute infarction in bilateral paramedian thalami and midbrain consistent with artery of percheron territory. MRA brain showed anterior and proximal posterior branches of the right middle cerebral artery (MCA) irregular luminal narrowing. All other vessels were unremarkable.

Several differentials were entertained during the initial stage including brain stem infarction, ocular myasthenia gravis or krait bite which went unnoticed. There was an initial difficulty of diagnosis because of patient's slight protracted history of 2 days and waxing and waning symptoms. Due to the diagnostic dilemma, urgent neurology referral was done after arrangement of MRI/MRA brain with NCS with repetitive nerve stimulation test (RNST). She was subsequently taken over to the neurology ward for further management since dates for MRI was delayed for 3 days.

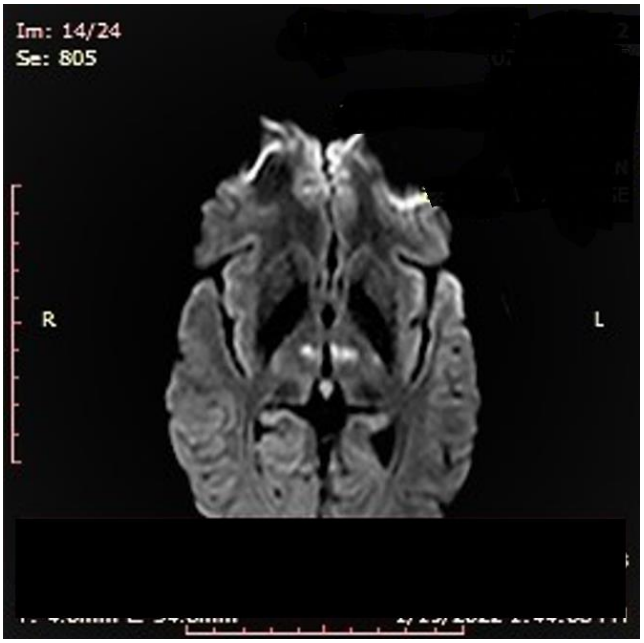


**Figure 2. MRI brain showing diffusion restriction in bilateral paramedian thalami and midbrain.**

**Therapeutic assessment and focus**

Thus, with above imaging report, a diagnosis of Artery of Percheron infarction was made. She was treated for an ischemic stroke and was started on aspirin 75 mg, clopidogrel 75 mg and atorvastatin 40 mg daily doses and clopidogrel was omitted after 3 weeks. Subsequently risk factor assessment and optimization were performed with 2 D echo found be significant for a left ventricular hypertrophy. Bilateral vertebral artery duplex was normal. Newly found diabetes was managed with oral hypoglycemic to achieve adequate control with arrangement of HbA1c and follow up was arranged at clinic level. Lipid profile showed LDL of 130 mg/dL with TG of 252 mg/dL. Considering the array of positive vascular risk factors, further testing for thrombophilias was deferred. She was reviewed in the clinic after discharge, but the clinical defects persisted with bilateral complete ptosis and presence of nystagmus. Memory impairment and hypersomnolence had settled. The vertical gaze palsy showed a slight

improvement. She could not perform her day-to-day activities from then onwards and was therefore discharged for home-based care with arrangement of social services.

## DISCUSSION

The thalamus is a deep cortical structure of the brain principally responsible for processing information pertaining to emotion, arousal, fine motor function and language, ocular movements cognitive function, sensory inputs and consciousness. The thalamus is subdivided into several functional nuclei, namely sensory, limbic associative, reticular, intralaminar, and effector nuclei. However, the arterial supply follows a different delineation subdivided into anterior, paramedian, inferolateral and posterior. As with any cortical domain, occlusion of the arterial supply of the thalamus can present with a spectrum of clinical presentations.

Vascular supply to the paramedian region of the thalamus is typically from perforating paramedian arteries which arise from the P1 segment of the PCA, and it shows several anatomical variations which contribute to confusing symptomatology at times. Described by Gerard Percheron in 1973, there are primarily 4 common variations of the paramedian artery and each gives rise to a different presenting symptomatology2. From these, the commonest is variant is type I, where both paramedian arteries originate from the PCA's of their respective sides. Type IIB is AOP, where the paramedian arteries start from a single P1 segment on one side, and subsequently divide and supply thalami of both sides and the rostral midbrain to a certain extent5 (Figure 1).

Thalamic infarctions comprise of 11% of all strokes as per a large study done in 19886. Comparing all thalamic infarctions, bithalamic infarctions showed a 0.6% prevalence in all ischemic stroke patients7. The prevalence of AOP in general population is still unknown. A small Cadaveric study done in 15 patients showed only one with AOP variation8. A case series on detection of commonest types of AOP infarction showed bilateral paramedian thalamic involvement with rostral midbrain infarction in a majority 43% of patients, additional anterior thalamic involvement in 14%, and bilateral paramedian thalami only involvement in 38%4. Bilateral paramedian infarctions typically demonstrate the triad of altered memory impairment, altered mentation and vertical gaze palsy9. Several other symptoms of combined bilateral paramedian involvement are dysfunction in arousal, learning deficits, decreased executive function, language deficits and visuo-spatial defects10. Rostral midbrain involvement gives rise to hypersomnolence, impaired responsiveness, hallucinations, behavioral abnormalities and associated oculomotor deficits with cortical blindness11. Differential diagnosis of bilateral thalamic lesions includes vasculitis, posterior reversible encephalopathy, infectious encephalitides, Korsakoff encephalopathy, osmotic myelinolysis, Creutzfeldt-Jakob disease and bilateral thalamic gliomas12. The treatment of AOP infarction is similar to any ischemic infarct including thrombolysis if indicated and prescription of antiplatelets, co-morbid management and arrangement of physiotherapy. The prognosis of bilateral thalamic infarcts is currently unknown. Several case series studies have conflicting reports of recovery thereby presenting a prognostic dilemma.

Our patient manifested two out of three features of bilateral paramedian infarction, namely, memory impairment and vertical gaze palsy. Additionally, hypersomnolence and nystagmus were present, indicating involvement of rostral midbrain. At the initial stage, the diagnosis was not straightforward with the ophthalmoplegia and diplopia, leading us in the direction of possible ocular myasthenia or krait bite in addition to midbrain/thalamic infarction. Evidence for the infarction was the memory impairment and hypersomnolence. The combination of the above symptoms was suggestive of artery of Percheron occlusion. Differential considered at onset were excluded by careful evaluation of history and examination with relevant investigations. Initial CT brain was normal and the MRI/MRA brain arranged within next few days confirmed the diagnosis of an AOP infarction. Delay in the MRI of 3 days might have contributed to the consolidation of neurological signs and poor clinical outcome. Her outcome was poor due to a combination of factors including inherent prognosis of AOP infarctions, delayed presentation, delayed

diagnosis and additional involvement of rostral midbrain.

This case highlights the need for updated knowledge on thalamic and midbrain syndromes to promote early detection and treatment to retain a favourable outcome. AOP infarction is an important rare differential to be considered in patients presenting with acute confusion, memory impairment and gaze palsy. Urgent MRI with MRA will confirm the definitive diagnosis and aid in the early treatment of these patients with a diagnostic conundrum. If detected in the therapeutic window, patients' therapeutic outcome may be improved.

## CONCLUSION

AOP infarctions are an infrequent subgroup of thalamic infarctions with an atypical and variable symptomatology, which might be misdiagnosed due to diagnostic difficulty and heterogeneity. High degree of suspicion, and early initiation of MRI brain will aid in the early diagnosis and prevention of morbidity in these patients, especially where initial CT brain is normal.

**Author declaration**

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Dr. TK Kannangara, Consultant physician, National Hospital, Kandy.

**Authors' contributions:**
Study concept and design: B.G.P; Drafting of the manuscript: W.G.M.D., U.H.J.P.D.; Study supervision: I.K.J.

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