**Bilateral thalamic stroke due to occlusion of the artery of Percheron; A case report and literature review**

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**CONFLICT OF INTEREST:-**

The authors would like to declare that they have no competing interests.

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**AUTHORS CONTRIBUTION:-**

1. **Eliz Achhami** : design of study, data collection, evidence collection, manuscript writing and revision, corresponding author.

2. **Seshkant lamichhane**: involved in patient management, data collection, design of study, manuscript writing and revision.

3. **Ashim Kandel**:- design of study, data collection, manuscript writing and revision

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**ETHICAL APPROVAL:-**

As case reports are exempt from ethical approval in our institution, our article which describes a case report does not require additional permissions from the Ethics committee.

**CONSENT:-**

Full written informed consent was obtained from the patient for publication of her case, clinical images, and radiographic images. A copy of written consent can be made available to the editor in chief of this journal upon request.

**DATA AVAILABILITY S TATEMENT:-**

All the data generated or analyzed during this study are included in the manuscript.

**Abstract:**

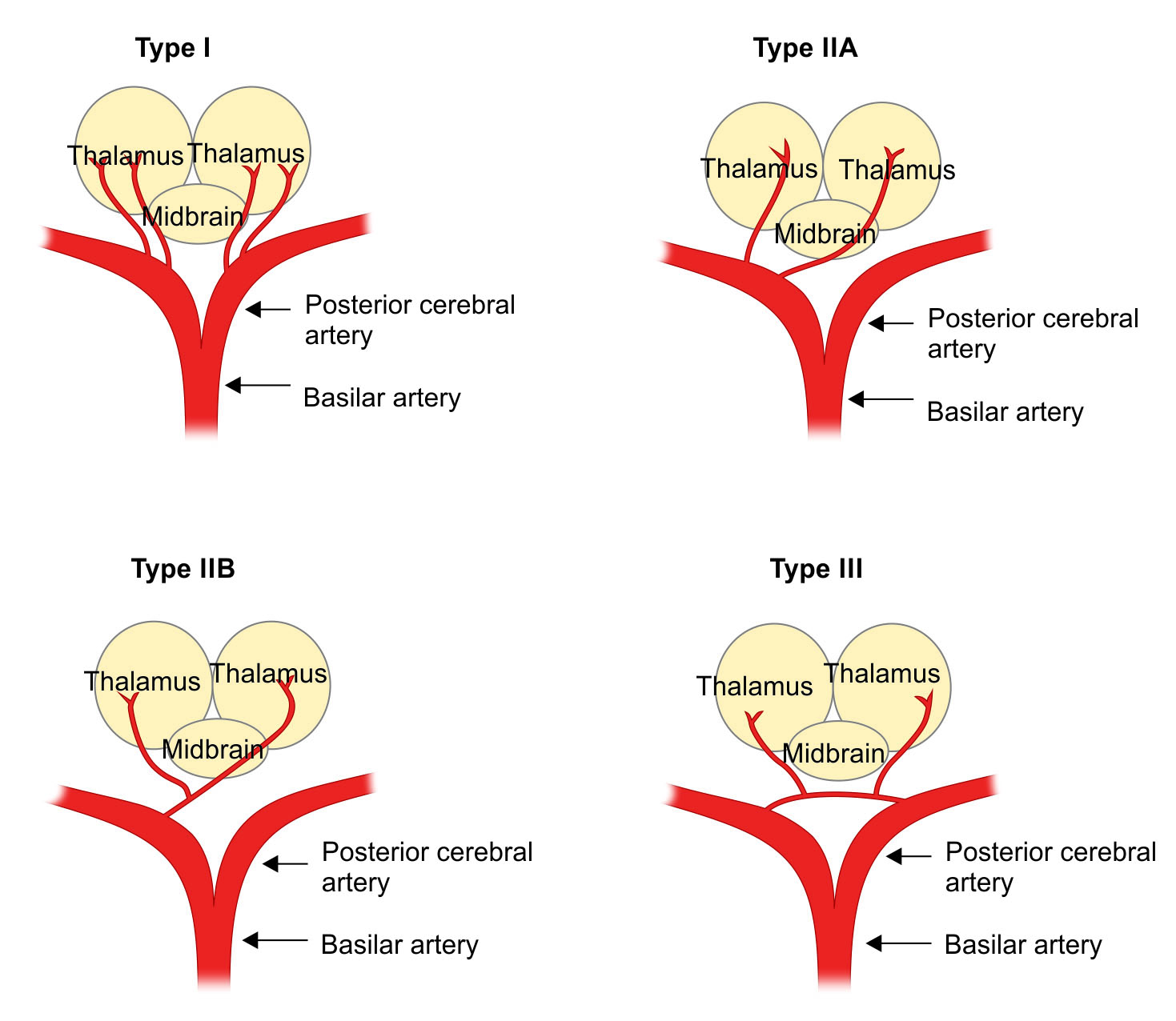
Bilateral thalamic infarction resulting from the occlusion of the artery of Percheron (AOP) is a rare cerebrovascular event with distinctive clinical presentations. This case report explores the intricate relationship between vascular anatomy, midbrain function, and clinical manifestations. A 48-year-old male farmer with a history of Diabetes mellitus presented with sudden-onset visual disturbances, diplopia, bilateral eyelid drooping, and loss of consciousness. Extensive evaluations, including advanced imaging techniques, led to the diagnosis of bilateral upper midbrain infarction involving AOP. This case underscores the complexity of neurovascular interactions, highlighting the importance of precise diagnosis and tailored management in addressing rare cerebrovascular conditions.

Keywords: Bilateral thalamic infarction, artery of Percheron, neurovascular complexity, midbrain function, clinical impact

**Background:-**

The blood supply to the thalami and midbrain comes from both the anterior (internal carotid arteries) and posterior (vertebro-basilar system) circulations, and various variations in this supply have been reported 1,2. Typically, the anterior circulation provides blood to the anteroinferior region of the thalami and midbrain through thalamoperforator arteries that arise from the posterior communicating arteries (PCAs). On the other hand, the posterior circulation supplies the medial aspects of the thalami and midbrain via branches originating from P1 segments and the lateral and superior aspects via branches arising from P2 segments of the posterior cerebral arteries (PCAs) 3. Coma caused by bilateral paramedian thalamic infarction resulting from the occlusion of the artery of Percheron (AOP) is an uncommon vascular condition, and its exact prevalence is not yet determined. Gérard Percheron first described the AOP in 1973 as an anatomical variant of the paramedian arteries originating from segment P1 of the posterior cerebral artery (PCA) 1. The suggested mechanism involves the occlusion of a single central thalamic perforating artery, which was previously described by Percheron 1 (Figure 1). The AOP variant is found in around 4% to 12% of the population 4. In two large stroke series studies, characteristic AOP infarct patterns accounted for 0.1% to 2% of all ischemic strokes, highlighting that this type of ischemic stroke is exceptionally rare 5.

In this case report, we present the clinical profile of a 48-year-old male farmer with a known history of Diabetes mellitus who presented with sudden-onset blurry vision, diplopia, bilateral drooping of eyelids, and loss of consciousness. The patient's initial management involved treatment for suspected meningioencephalitis, given the presence of fever and unresponsiveness. However, further investigations, including brain MRI and MRA, led to the diagnosis of bilateral upper midbrain infarction involving the artery of Percheron.



**Figure No. 1 :- A diagram illustrating the four variants of the arterial supply to the Thalamus**

**Case Presentaiton :-**

A 48-year-old male, a farmer by profession, with a known history of Diabetes mellitus for 5 years and on metformin 500 mg twice a day, presented with a sudden onset of blurry vision, diplopia (double vision), and bilateral drooping of eyelids and loss of consciousness while he was working in the field. He was immediately taken to the local hospital where he had a fever of 101 degrees Fahrenheit. At the local hospital, he received intravenous fluids, intravenous paracetamol, and intravenous ceftriaxone. Due to the severity of his condition, he was subsequently referred to a tertiary care center for further management.

Upon arrival at the tertiary care center, the patient had a Glasgow Coma Scale (GCS) score of E2V3M4, indicating a severe neurological impairment. His blood pressure was recorded at 130/70 mmHg and his pulse was elevated at 110 beats per minute. The respiratory rate was 20 breaths per minute, and his temperature had decreased to 99 degrees Fahrenheit. While his oxygen saturation was 95 percent in room air, his bilateral eyes exhibited lateral deviation, and the pupils were non-reactive, measuring 5mm in size. However, he did maintain intact cough and gag reflexes, and neck rigidity was notably absent. The patient responded with limb movements upon experiencing painful stimuli. During the cardiovascular, respiratory, and abdominal examinations, no abnormalities were note.

The patient underwent a non-contrast CT scan of the head, which did not show any significant findings. However, due to the presence of fever and unresponsiveness, the medical team decided to initiate treatment for meningioencephalitis. Intravenous ceftriaxone, intravenous vancomycin, intravenous acyclovir, and intravenous dexamethasone were administered in line with this suspected diagnosis. Subsequently, a lumbar puncture was performed, and cerebrospinal fluid (CSF) analysis revealed a total cell count of 6 with 100 percent lymphocytes, glucose level of 88 mg/dL, protein level of 42 mg/dL, and an ADA (adenosine deaminase) level of 7.5. Despite extensive testing, including CSF gram stain, culture sensitivity, and gene xpert analysis, no specific causative agent was identified. PCR testing for herpes simplex 1 and 2 in the CSF also returned negative results. The patient's laboratory findings at the time of admission and at the time of discharge are summarized in Table 1.

An MRI of the brain was conducted, revealing altered T2/FLAIR signal intensity lesions in bilateral thalamus, including the midbrain, with restricted diffusion and blooming artifact (Figure 2). Further assessment through MRA demonstrated non-visualization of the right P1 segment, and it was noted that the P1 segment was arising from the right posterior communicating artery (Pcom) (Figure 3). To investigate further, serology for dengue and Japanese encephalitis was performed, but the results came back negative. Considering the MRI findings, there was a suspicion of bilateral upper midbrain infarction involving the artery of Percheron.

Because of more than 24 hours of documented stroke diagnosis since he was known at his baseline, the patient was not a candidate for tissue plasminogen activating factor (tPA). The neurosurgery team recommended conservative management. The patient continued to improve. Within the first 48 hours, the patient was confused but awake and responsive to minimal commands. In light of the patient's condition remaining stable with a GCS of E3V4M4 over three weeks of treatment, a decision was made to discharge the patient for home care and management. Medications such as tab aspirin 75 mg and tab rosuvastatin 20 mg were prescribed, likely to manage the stroke-related aspects of the condition. After one month of follow-up, the patient continues to experience difficulty in opening his eyes spontaneously. However, they are showing positive signs of responsiveness by communicating verbally and demonstrating spontaneous movement in all their limbs.

| **Parameter** | **Value** | **Reference Range** |
| --- | --- | --- |
| Tlc (Total Leukocyte Count) | 10400 | 4000 - 11000 / μL |
| Neutrophil | 83 | 40 - 75% |
| Lymphocytes | 13 | 20 - 40% |
| Monocytes | 3 | 2 - 8% |
| Eosinophils | 2 | 0 - 6% |
| Platelets | 245000 | 150000 - 450000 / μL |
| Hemoglobin | 13 | 12 - 16 g/dL |
| PT/INR | 13/1 | 0.8 - 1.2 |
| Urea (ur) | 90 | 20 - 40 mg/dL |
| Creatinine (cr) | 0.7 | 0.6 - 1.2 mg/dL |
| Sodium (Na) | 146 | 135 - 145 mmol/L |
| Potassium (K) | 4 | 3.5 - 5.1 mmol/L |
| Total Bilirubin (TB) | 1 | 0.2 - 1.2 mg/dL |
| Direct Bilirubin (DB) | 0.3 | 0 - 0.3 mg/dL |
| AST (Aspartate Aminotransferase) | 20 | 10 - 40 U/L |
| ALT (Alanine Aminotransferase) | 28 | 7 - 56 U/L |
| ALP (Alkaline Phosphatase) | 59 | 38 - 126 U/L |
| Total Protein | 5.4 | 6.0 - 8.3 g/dL |
| Albumin | 2.7 | 3.4 - 5.4 g/dL |
| Free T3 (fT3) | 4.37 | 2.3 - 4.2 pg/mL |
| Free T4 (fT4) | 14 | 9 - 23 pmol/L |
| TSH (Thyroid Stimulating Hormone) | 1.12 | 0.4 - 4.0 µIU/mL |
| Calcium (Ca) | 8.9 | 8.5 - 10.5 mg/dL |
| Magnesium | 2.04 | 1.7 - 2.3 mg/dL |
| VDRL (Venereal Disease Research Laboratory) | Non Reactive | Non Reactive |
| Hepatitis B | Non Reactive | Non Reactive |
| Hepatitis C | Non Reactive | Non Reactive |
| HIV | Non Reactive | Non Reactive |
| HbA1c | 7 | 4.0 - 5.6% |
| Dengue Serology | Negative | Negative |
| Japanese Encephalitis Serology | Negative | Negative |
| HSV1/2 PCR | Negative | Negative |

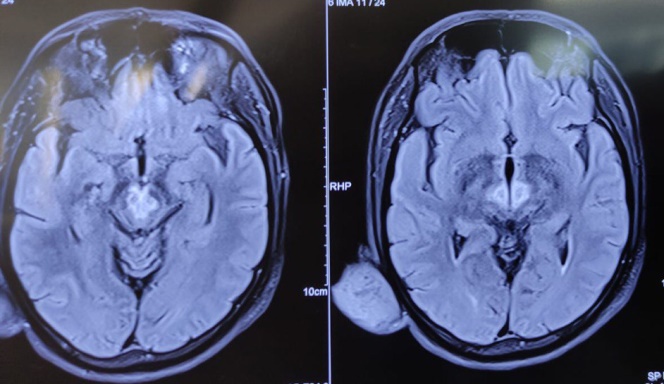
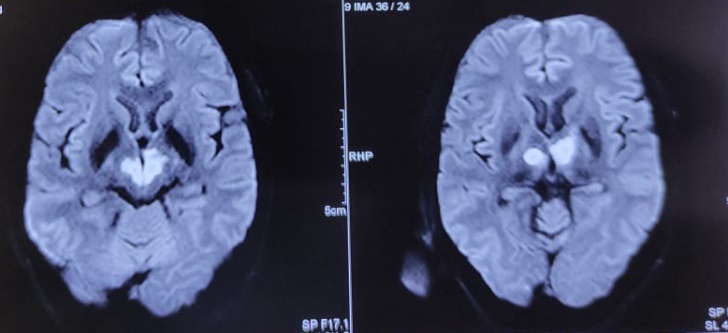


Figure 2:- MRI of the brain revealing T2/FLAIR signal intensity lesions in bilateral thalamus, including the midbrain.

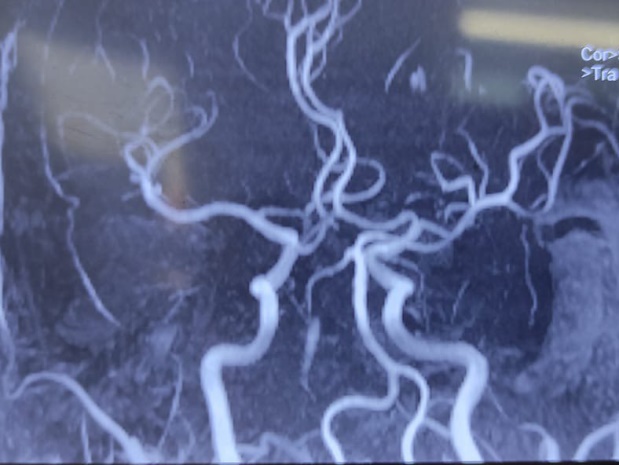


Figure 3:- MRA demonstrating non-visualization of the right P1 segment, and P1 segment arising from the right posterior communicating artery

**Discussion and Conclusions:-**

Bilateral thalamic infarction due to AOP occlusion is an uncommon cerebrovascular event with distinctive clinical manifestations. In this case, the patient, a 48-year-old male farmer with a history of Diabetes mellitus, experienced sudden-onset blurry vision, diplopia, bilateral eyelid drooping, and loss of consciousness while working in the field. Upon examination at the tertiary care center, the patient displayed severe neurological impairment, including unconsciousness, non-reactive pupils, and lateral deviation of the eyes. These clinical features, along with the MRI findings of altered T2/FLAIR signal intensity lesions in bilateral thalami and the midbrain, strongly suggested bilateral thalamic infarction resulting from artery of Percheron occlusion.

The patient's clinical presentation in this case was characterized by sudden-onset blurry vision, diplopia, bilateral ptosis, and loss of consciousness. The presence of lateral deviation in the bilateral eyes and non-reactive pupils pointed towards a significant midbrain involvement. The absence of neck rigidity was notable and helped distinguish this case from typical presentations of infectious causes, prompting further investigations into cerebrovascular etiologies. The clinical findings of non-reactive pupils and lateral eye deviation also align with midbrain involvement. The midbrain's role in regulating pupillary responses to light and eye movements is well-documented. Non-reactive pupils and abnormal eye positioning are consistent with the broader neurological disruption associated with midbrain infarctions.

Sequelae of bilateral infarction in the paramedian thalamic region have been reported to include decreased levels of consciousness, alterations in mental status, apathy, memory loss, difficulty with language (aphasia), speech difficulties (dysarthria), motor deficits, and disinhibited behaviors 6–8. If the entire midbrain is affected, this leads to mesencephalothalamic or thalamopeduncular syndrome, which can include other oculomotor disorders and the involvement of long motor pathways 9–12.

The diagnosis of AOP occlusion relies on cerebral imaging studies, primarily MRI, since CT findings may appear normal 13. To identify AOP infarct early on, FLAIR and diffusion-weighted imaging are the preferred imaging techniques 4,8. Although an initial MRI brain scan may be normal, it cannot exclude AOP ischemic infarcts 14. Hence, it is advisable to consider a repeat MRI focused on the vertebrobasilar territory 15. While CT may be more accessible initially, MRI is essential and should be performed either as the primary or secondary imaging study. Ischemic lesions of the medial parts of both thalami, with or without rostral mesencephalic involvement, are observed 4. The midbrain 'V' sign, which appears as a high-intensity signal on axial FLAIR and diffusion-weighted images along the pial surface of the midbrain in the interpeduncular fossa, has been reported in 67% of patients with AOP occlusion 4. In our case the MRI of the brain revealed altered T2/FLAIR signal intensity lesions in bilateral thalamus, including the midbrain, with restricted diffusion and blooming artifact. The non-visualization of the right P1 segment and its origin from the right posterior communicating artery (Pcom) raised suspicion of bilateral upper midbrain infarction involving the artery of Percheron.

The main differential diagnosis involves bilateral thalamic venous infarction that complicates internal cerebral vein thrombosis 13,16,17. Although bilateral thalamic infarction can be part of the top of the basilar syndrome 18, it is uncommon to find isolated bilateral paramedian thalamic infarction 18. In our case, the working diagnosis was stroke; however, other less probable conditions could also lead to bilateral thalamic lesions. These conditions encompass thiamine deficiency 19, cerebral lupus 20, abscesses caused by fungi or toxoplasmosis 21, cerebral syphilitic gumma 22, and tumors 23. Additionally, in children, bilateral thalamic necrosis has been associated with influenza 24.

The thalamic infarction can be linked to venous sinus thrombosis, resulting from occlusion of internal cerebral veins 25. Symptoms include headache, vomiting, and papilledema, leading to infarction with seizures, focal neurological deficits, and aphasia 25. Wernicke’s encephalopathy is also included in the differential diagnosis because it results in lesions in bilateral thalami, periaqueductal gray tectal plate, dorsal medulla, and mammillary bodies 26. Infections and osmotic myelinosis may be included in the differential diagnosis, as well 25.

Artery of Percheron (AOP) infarcts should be considered as a possible diagnosis when dealing with altered mental status in elderly patients. According to a study, memory impairment was observed in 58% of patients, while coma was present in 42%, confusion in 53%, and vertical gaze palsy in 65% of patients 5.

Thalamic dementia results from bilateral thalamic infarcts, not unilateral ones. It leads to memory impairment, confusion, and coma due to disconnection between the thalamus and cortex caused by a paramedian thalamic infarct 1.

The standard treatment for an acute ischemic stroke depends on factors such as timing, lesion location, and contraindications to thrombolytics. For cases involving proximal cerebral artery occlusion, the recommended approach is the administration of recombinant tPA within 4.5 hours of onset, combined with mechanical thrombectomy within 6 hours 27. In contrast, AOP represents a distal artery of the posterior circulation with a small diameter, and information on the efficacy of mechanical removal is challenging to find. Due to its size, endovascular revascularization is rarely considered as an option for AOP infarcts, as it is not easily visualized 28.

**Conclusion:-**

In conclusion, this rare case of bilateral thalamic infarction resulting from artery of Percheron occlusion emphasizes the intricate relationship between vascular anatomy, midbrain function, and clinical manifestations. The patient's abrupt onset of visual disturbances, diplopia, and eyelid drooping underscored the involvement of critical midbrain structures, notably the third cranial nerve. Advanced imaging techniques played a crucial role in diagnosis, revealing characteristic MRI findings. As such, this case serves as a reminder of the complexity of cerebrovascular events and the necessity for vigilant clinical assessment, leading to accurate diagnosis and tailored management strategies

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