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Wake-Up Stroke: Clinical Challenges in the Artery of Percheron

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Abstract: A stroke involving the artery of Percheron is an infrequent condition arising from an anatomical variant in the vascular supply of the thalamus and midbrain. This particular vascular configuration can lead to bilateral thalamic infarctions, characterized by significant neurological impairment, including altered consciousness, cognitive deficits, and motor dysfunction. Identifying this pathology is challenging due to its atypical clinical presentation. This case report describes a wake-up ischemic stroke associated with the artery of Percheron. Its purpose is to analyze the diagnostic challenges, therapeutic interventions, and management strategies employed in the emergency department setting, highlighting the educational and clinical implications that such events contribute to the medical literature.

Keywords: *Percheron Artery, Wake-Up Stroke, Bilateral Thalamus Infarcts.*

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I. INTRODUCTION

An ischemic stroke in the artery of Percheron is rare, accounting for 0.1–0.3% of all ischemic strokes and between 4–18% of thalamic ischemic strokes (1). To understand a Percheron stroke, it is important to review the usual vascular supply of the thalamus. The thalamus and midbrain receive blood flow from penetrating branches of the posterior cerebral arteries and communicating arteries (2,3). Thalamic blood supply is divided into four main territories: anterior, paramedian, posterior, and inferolateral (4).

The paramedian territory is supplied by the thalamic paramedian arteries, which originate from the P1 segment of the posterior cerebral artery (5). Their configuration and origin vary and have been classified into three subtypes (Figure 1):

- Type I: The most common variant, in which each thalamic paramedian artery arises symmetrically from the P1 segment of each posterior cerebral artery.
- Type II: An asymmetric arrangement with two sub-variants:

✓ Type IIA: The thalamic paramedian arteries originate from the P1 segment of only one posterior cerebral artery.
✓ Type IIB or artery of Percheron: Present in approximately 4–12% of the population (5,6). It arises from a single P1 segment with a common trunk that then bifurcates into two thalamic paramedian arteries. This single trunk supplies both paramedian thalami and the rostral midbrain.

- Type III: As in Type I, each thalamic paramedian artery originates from its respective P1 segment of the posterior cerebral arteries, but there is a communicating arcade that gives rise to the paramedian thalamic branches (5)

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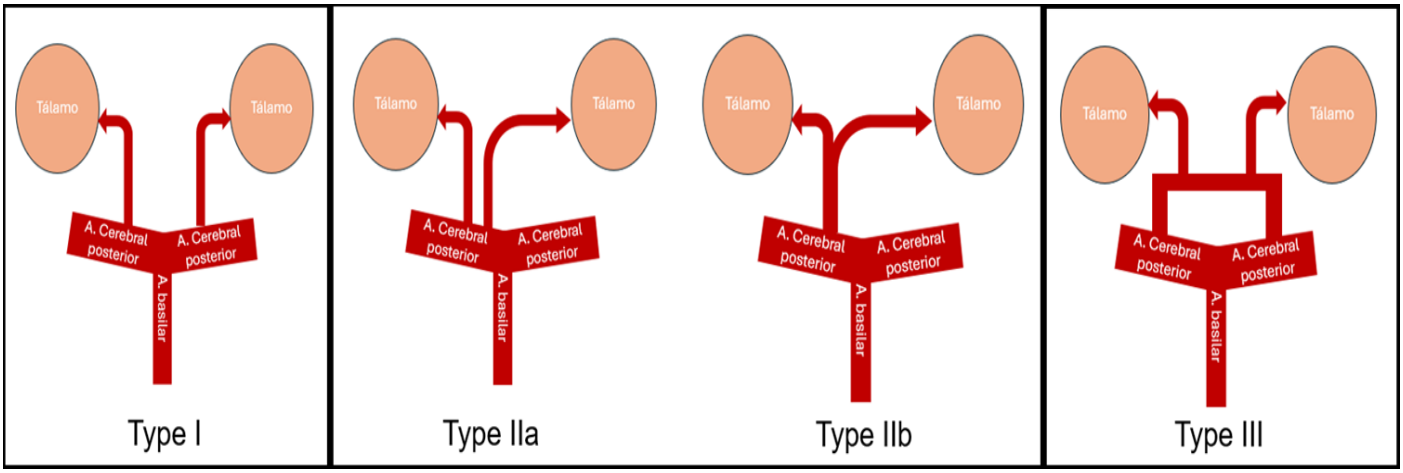

Fig 1 Anatomical Variants of Paramedian Thalamic Vascularization.

II. CASE DESCRIPTION

A 69-year-old man from Bogotá, with a history of poorly controlled hypertension, active smoking (20 pack-years), and chronic lung disease, was brought to the emergency department. A relative found him in bed around 11:00 a.m., responding only minimally to verbal stimuli and exhibiting dysarthria. The patient had last been seen at his neurological baseline at approximately 8:00 p.m. the previous night.

On arrival, his blood pressure was 180/103 mmHg, heart rate 92 bpm, respiratory rate 15 breaths/min, and oxygen saturation 52% in room air. Oxygen therapy via nasal cannula at 2 L/min was initiated, achieving saturations above 90%. Neurologically, he was somnolent, had dysarthria, upward gaze deviation with vertical ocular palsy, but preserved bilateral pupillary reactivity. He was unable to stand or walk (no bipedal support), though no paresis, plegia, or sensory deficits were noted in the face or limbs. His NIH Stroke Scale (NIHSS) score was 2, and Glasgow Coma Scale (GCS) was 14/15.

Suspecting a cerebrovascular accident (CVA), a non-contrast cranial computed tomography (CT) scan was performed (Figure 1), showing no significant findings. Laboratory tests revealed the following: white blood cell count 4,930/µL (neutrophils 3,350/µL, 67.97%), hemoglobin 20.44 g/dL, hematocrit 62.42%, platelets 197,800/µL, prothrombin time 11.8 s, INR 1.05, and partial thromboplastin time 29.46 s. Renal function showed creatinine of 0.78 mg/dL and blood urea nitrogen of 15.4 mg/dL; electrolytes were within normal limits (Na+ 140.3 mEq/L, K+ 4.6 mEq/L, Cl– 104.23 mEq/L). Liver function tests revealed total bilirubin of 0.71 mg/dL (direct 0.26 mg/dL, indirect 0.45 mg/dL), ALT 16.4 U/L, AST 21.9 U/L, and alkaline phosphatase 99.3 U/L. Arterial blood gases showed pH 7.33, PaCO2 44.4 mmHg, PaO2 63 mmHg, HCO3– 23.5 mEq/L, base excess –2.6, and a PaO2/FiO2 ratio (P/F ratio) of 175.

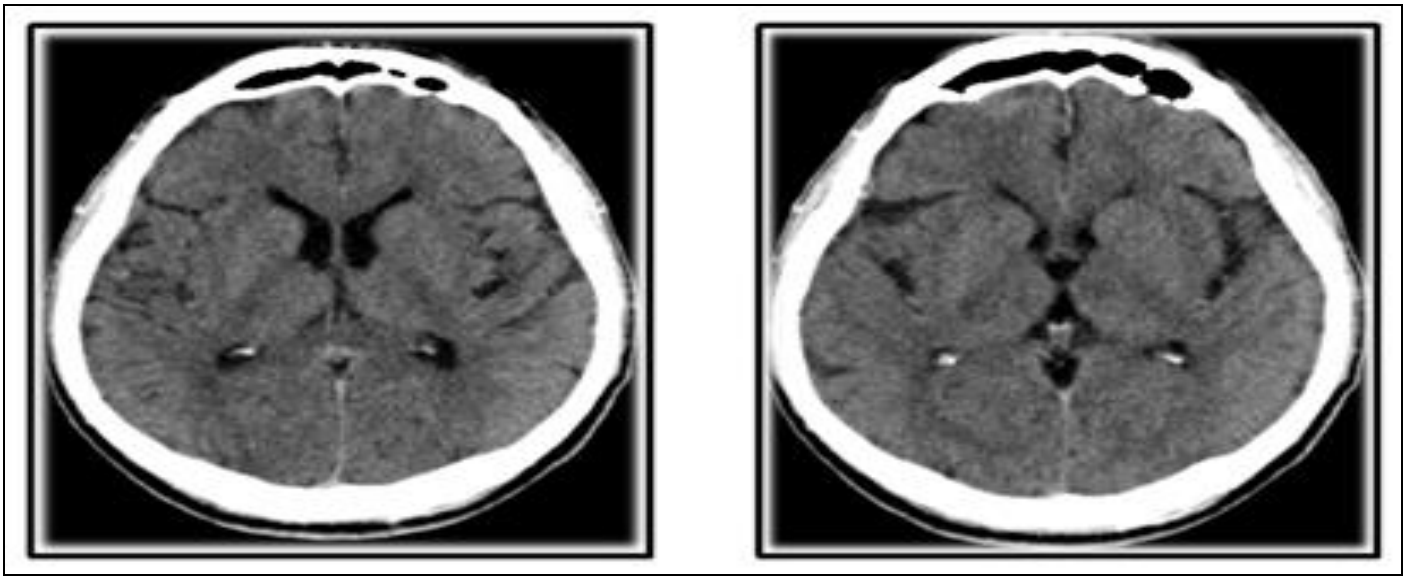

Fig 1 Non-Contrast Cranial CT Scan showing no Hemorrhagic or Ischemic Lesions.

Given the high clinical suspicion, a brain magnetic resonance imaging (MRI) was performed, revealing hyperintense lesions on DWI (Diffusion Weighted Imaging), FLAIR, and T2 sequences (Figure 2), with diffusion restriction involving the medial bilateral thalamic regions. These findings were consistent with late hyperacute ischemia, precluding the use of pharmacological reperfusion therapy.

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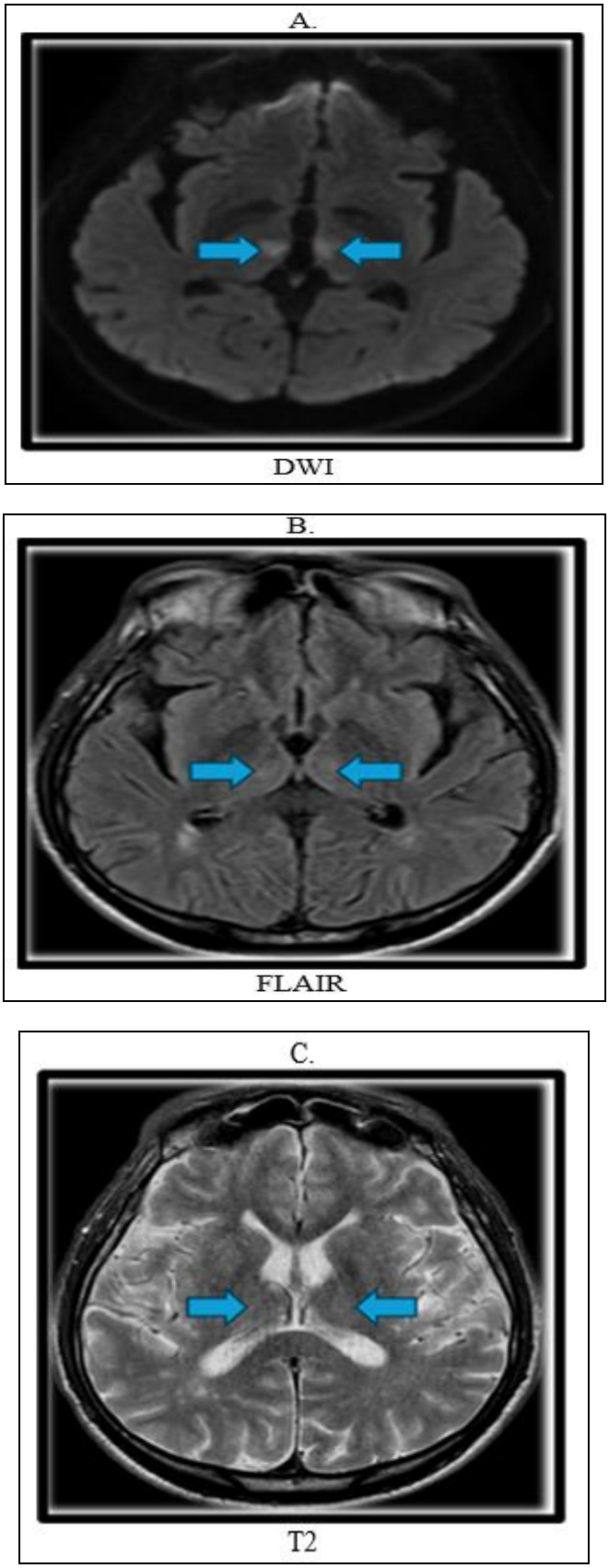

Fig 2 A. (DWI sequence) demonstrating bilateral paramedian hyperintensities at the thalamic level. B. (FLAIR) and C. (T2) showing incipient changes in the same regions, compatible with the onset of vasogenic edema. These findings suggest a late hyperacute ischemic event in the artery of Percheron territory.

The patient remained under continuous monitoring in the resuscitation area and received labetalol to control elevated blood pressure. Approximately six hours after admission, he experienced a sudden deterioration in consciousness, prompting airway protection with endotracheal intubation and transfer to the intensive care unit.

III. DISCUSSION

Due to its rarity, a Percheron stroke can be easily underdiagnosed or overlooked by medical staff in the emergency setting. Its clinical presentation is often atypical, usually associated with seven main patterns (6):

- Alterations in level of consciousness, ranging from irritability to stupor or coma.
- Behavioral and memory changes, ranging from confusion and disinhibition to eating disorders.
- Aphasia and/or dysarthria.
- Motor deficits, including cranial nerve involvement or limb weakness.
- Abnormal eye movements, such as vertical or horizontal gaze palsies or non-reactive pupils.
- Cerebellar signs like dysmetria or ataxia.
- Other nonspecific manifestations, including seizures, hypersomnia, and hyperthermia.

A comprehensive medical history and detailed neurological examination are critical in the emergency department when a stroke is suspected. Nonetheless, definitive diagnosis and therapeutic decision-making require integrating multiple neuroimaging modalities. A non-contrast CT scan of the head remains the initial method of choice to exclude intracranial hemorrhage; however, relying solely on CT is insufficient for definitive management (7).

Perfusion CT can provide dynamic information on core infarction and penumbra. Still, the absence of findings on perfusion imaging does not rule out the possibility of artery of Percheron stroke (6,7). Owing to the extremely small caliber of this artery, conventional angiographic techniques, including MR angiography, often do not enable direct visualization, making it difficult to detect occlusions or stenosis at this level (8).

In the specific context of wake-up strokes, accurately determining when the patient was last seen symptom-free is vital for guiding therapy. However, the exact optimal time window for safely administering fibrinolytic therapy in these cases remains undetermined. A prospective study by Manawadu et al. evaluated patients with time intervals of 4.5 to 12 hours from their last asymptomatic observation, who subsequently received thrombolysis. The results showed no significant differences in modified Rankin Scale scores of 0–2 at 90 days or in symptomatic intracerebral hemorrhage rates compared to patients treated following standard guidelines (9).

Here, MRI plays a central role, enabling consideration of extended thrombolysis windows and allowing for early fibrinolytic intervention. Early signs of cellular edema,

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identifiable via DWI sequences and apparent diffusion coefficient (ADC) values, as well as the absence of changes (mismatch) between FLAIR and perfusion-weighted imaging (PWI), are markers of vasogenic edema and potentially salvageable low-flow territories. These MRI findings are particularly beneficial when symptom onset time is unclear, as with wake-up strokes (7,10).

In artery of Percheron stroke specifically, there is significant interindividual variability regarding lesion symmetry and extent. This variability is attributed to differences in vascular patterns and in the number of involved branches. Nevertheless, a characteristic pattern typically involves bilateral paramedian thalamic lesions that show hyperintensity on DWI and FLAIR with associated diffusion restriction on ADC maps (8). Differential diagnoses for bilateral thalamic lesions include top-of-the-basilar syndrome, venous sinus thrombosis, Wernicke's encephalopathy, neuroinfections, and osmotic demyelination (6).

Management of artery of Percheron stroke follows established guidelines for ischemic stroke, including pharmacological fibrinolysis with alteplase within 4.5 hours of symptom onset and consideration of mechanical thrombectomy in selected patients, based on clinical and imaging criteria that justify extending the treatment window (11). Notably, it is crucial to identify clinically significant neurological deficits that, despite low NIHSS scores, result in substantial functional limitations. Even if such deficits are the sole clinical manifestations, they should not be viewed as an exclusion criterion for fibrinolytic therapy.

IV. CONCLUSIONS

This review of a stroke in the artery of Percheron, together with the supporting literature, underscores the diagnostic and therapeutic complexity of this rare but potentially debilitating condition. Anatomical and clinical variability, as well as subtle or atypical neurological signs, can hinder recognition in the emergency department. Advanced neuroimaging—particularly MRI with DWI, ADC, FLAIR, and PWI sequences—is essential for delineating the ischemic core, identifying extended therapeutic windows, and guiding decisions on thrombolysis, especially in uncertain scenarios such as wake-up strokes.

Despite the lack of precise temporal criteria and the difficulty in directly visualizing the artery of Percheron using conventional angiographic studies, adherence to established protocols for ischemic stroke, recognition of functionally significant deficits, and a comprehensive therapeutic approach—including fibrinolysis and mechanical thrombectomy in selected patients—can improve clinical outcomes and quality of life.

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