Enfarte da Artéria de Percheron: Caso Clínico

Artery of Percheron Infarction: Case Report

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Resumo:

A artéria de Percheron (AOP) é uma variante normal que irriga o tálamo. Os enfartes da AOP podem originar um espectro alargado de apresentações clínicas invulgares. O seu diagnóstico é dificultado pela conjunção de dois factores: falta de sensibilidade da tomografia computadorizada e falta de conhecimento desta entidade motivada pela sua raridade. Descrevemos o caso de uma doente com coma por enfarte da AOP. Pretendemos salientar a importância de considerar esta entidade no diagnóstico diferencial de doentes com défices neurológicos vagos de origem indeterminada. Reforçamos ainda a necessidade de baixar o limiar para a realização de ressonância magnética com o objetivo de facilitar o diagnóstico e diminuir a morbilidade associada.

Palavras-chave: Artéria Cerebral Posterior/diagnóstico por imagem; Enfarte Cerebral/diagnóstico por imagem; Ressonância magnética.

Abstract:

The artery of Percheron (AOP) is a normal variant that supplies the thalamus. AOP infarcts may originate a spectrum of unusual clinical presentations. The diagnosis is difficult due to the lack of sensitivity of detection by CT scan and to the lack of acknowledgment of this clinical entity. We describe the case of a patient who presented with unresponsiveness due to an AOP infarct. Our report serves as a reminder to consider AOP infarct as differential diagnosis when assessing patients with unspecific neurological deficits of unclear origin. Further we reinforce the claim for a low threshold for MRI in patients presenting acutely with otherwise unexplainable neurological symptoms to facilitate diagnosis and decrease patient morbidity.

Keywords: *Cerebral Infarction/diagnostic imaging; Magnetic Resonance Imaging; Posterior Cerebral Artery/diagnostic imaging.*

Introduction

The paramedian territories of the thalami are supplied by perforating thalamic arteries of posterior circulation through 1 of 4 variants (Fig. 1). Variant I is the most common with perforating branches arising from both posterior cerebral arteries (PCAs) accounting for about 55% of cases. In variant IIa the left P1 segment is the source of both paramedian arteries (12% of cases) while in variant IIb, perforating arteries derive from the artery of Percheron (AOP) that arises from a part of the posterior cerebral artery (21% of cases). Finally, in variant III there is an arterial arc bridging PCAs together (12% of cases).[1-4]

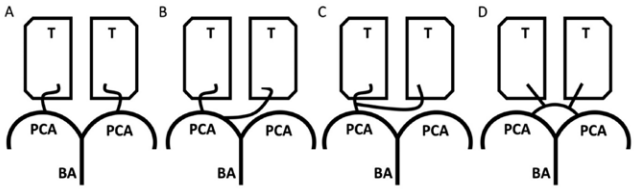

Figure 1: Paramedian thalamic artery variants. A – Type I, most frequent variant anatomy with perforating branches supplying the thalami (T) arising from the both posterior cerebral arteries (PCAs) that derive from the basilar artery (BA); B – Type IIa, both paramedian arteries originate from the P1 segment of the left PCA; C – Type IIb, the artery of Percheron (AOP) originates unilaterally from the P1 segment of PCA and then bifurcates, supplying the bilateral paramedian thalamus; D – Type III, an arterial arc connects the left and right P1 segments of PCA and gives rise to the paramedian arteries.
Adapted from Amin OS, *et al.* Bilateral infarction of paramedian thalami: a report of two cases of artery of Percheron occlusion and review of the literature. BMJ Case Rep. 2011 ;2011:bcr0920103304.[1]

AOP is an uncommon single-artery pathology that can affect bilateral thalamic structures. Bilateral thalamic infarction is rare representing around 0.6% of stroke cases and 22%-35% of thalamic infarcts.[5-6]

More frequent ischemic strokes such as those affecting the middle cerebral artery, produce predictable focal neurological deficits and clinical syndromes. In contrast, AOP infarcts lead to a spectrum of clinical presentations that include altered mental status, from transient or episodic loss of consciousness to coma, memory impairment, psychosis, aphasia, dysarthria, and oculomotor dysfunction.[7-13] The unusual presentations (symptoms and signs) are one of the reasons why AOP strokes are often missed. Other contributors are the difficulty of detecting the infarct on primary imaging and also the lack of acknowledgment of this clinical entity given its rarity.[14]

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DOI:10.24950/CC/214/20/2/2021

Case Report

A 93-year-old Portuguese female, partially dependent (Katz 3) due to osteoarthritis with no other medical history or co-morbidities was found unresponsive in the morning, eight hours after her last witnessed normal baseline. Her family was unable to awake her, prompting them to call emergency services that transported her to the hospital.

Remaining history and review of systems were limited by the patient's condition. Physical exam on arrival to the emergency department revealed a comatose patient. She had minimally reactive, positioned at midline and symmetrical 4 mm pupils with no abnormal ocular movements and preserved corneal reflexes. Deep tendon reflexes were preserved, and muscle tone was decreased, although the patient was able to move her extremities in response to painful stimuli with apparent decrease in mobility affecting her right side. Plantar responses were flexor on both sides. The respiratory pattern was normal and cardiovascular and other systemic examination was unremarkable.

No pharmacological context that could contribute to the clinical picture was identified. Nonetheless, a putative opioid or benzodiazepine intoxication was excluded through naloxone and flumazenil administration without response.

Biochemical laboratory tests and blood counts were normal, and electrocardiogram was unremarkable. The initial computed tomography (CT) scan of the head without contrast documented previous right lacunar infarct and microvascular ischemic disease. No acute intracranial pathology was found (Fig. 2a).

Based on the vague clinical signs presented, poor focal findings, and lack of evidence supporting acute ischemia on imaging study, there was low suspicion for acute stroke and the patient was admitted for clinical surveillance and for diagnostic workup. Forty-eight hours after admission, the CT scan was repeated allowing the identification of hypodensities visible on both thalami, more evident on the left side extending to mesencephalic region. Twenty-four hours later diffusion-weighted magnetic resonance imaging (MRI) was performed revealing acute infarcts in the bilateral thalami extending toward the ventral midbrain compatible with an acute ischemic lesion affecting the AOP territory with evidence of haemorrhagic transformation (Fig.s 2b and 2c). Echocardiography, Holter and carotid ultrasound showed no clues regarding infarction aetiology.

Throughout the hospital stay, the patient maintained the comatose state and was transferred to a continuing care unit.

Discussion

The thalamus is a bilateral structure composed of several nuclei serving as a "relay centre" for a broad spectrum of neurological functions that include movement, arousal, cognition, behaviour, and emotion. This justifies the many possible presentations of an AOP stroke and reinforces the need for awareness of those. The clinical presentation may vary widely from mild symptoms such as dizziness and confusion to severe signs like coma.[8,15] Classic manifestations include altered level of consciousness, vertical gaze palsy, and cognitive impairment although classic signs of a stroke or focal lesion, as aphasia, dysarthria, and oculomotor disturbance may also be present.[15] Table 1 summarizes the common presentations seen in AOP infarction. Considering the anatomic correlates proposed in the literature, our patient's decreased level of arousal probably is due to intralaminar nuclei damage given its input from the brainstem ascending reticular activator mechanism, which mediates alert consciousness.[14]

The literature reports similar aetiologies as for other perforating arteries infarctions. In a study by Xu *et al*, the most frequent cause of AOP infarction was small vessel disease, followed by cardioembolism.[15]

CT imaging frequently misses abnormalities in acute AOP

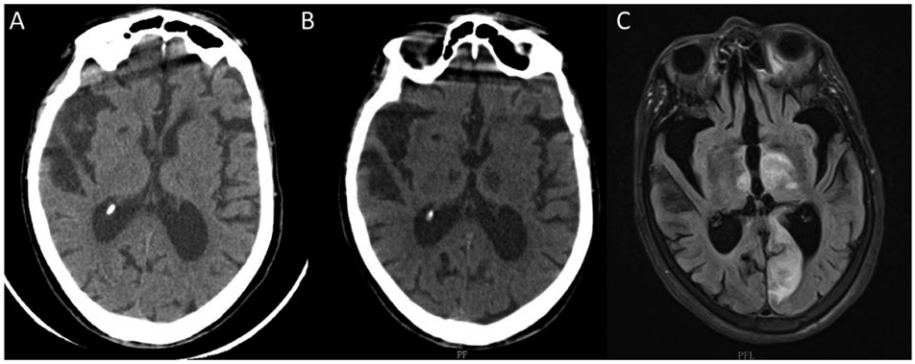

Figure 2: Brain imaging across time. A) Head computed tomography (CT) scan at admission showing no acute intracranial pathology documenting previous right lacunar infarct and microvascular ischemic disease (figure 2a). B) CT at 48 hours after admission demonstrating hypodensities visible on both thalami, more evident on the left side extending to mesencephalic region. C) T2-weighted brain MRI at 24 hours after admission picturing subacute bilateral infarcts affecting both thalami and mesencephalon most evident on the left along with subacute infarct affecting the territory of left posterior cerebral artery with evidence of hemorrhagic transformation.

Table 1: AOP infarcts presentations in the literature.

Category	Clinical manifestation	Refs
Mental status changes	Somnolence, stupor, and coma.	(3,8,12,15)
Behavior and memory impairment	Confusion, delirium, psychosis, apathy, hyperphagia, and pseudobulbar affect.	(9,10)
Ocular movement abnormalities	Horizontal and vertical gaze paresis with or without pupillary involvement.	(3)
Motor deficits	Paresis or paralysis affecting the face or limbs.	(15)
Cerebellar alterations	Ataxia and dysmetria.	(12)
Speech abnormalities	Aphasia, dysarthria and slurred speech.	(15)
Other nonspecific alterations	Drowsiness, tremors, seizures, hyperthermia and bradycardia.	(7,15)

infarction as was the case for our patient at admission. The same happens with CT angiography since occlusions in such small calibre arteries are usually not identified. MRI, on the contrary, has been shown to detect all AOP stroke cases in a small series.[15] As result, many patients with AOP stroke remain undiagnosed several hours/days until MRI is performed. The vague presentation of the case presented herein and negative CT scan at admission concurred to an initial low suspicion level of a cerebrovascular event. Added to the unavailability of MRI at the emergency department, the diagnosis was delayed compromising both treatment and prognosis. Early detection and intervention significantly impact functional outcomes for patients.

Our report serves as a reminder to consider AOP infarct as differential diagnosis when assessing patients with unspecific neurological deficits of unclear origin, namely unexplained depressed level of consciousness. A low threshold for MRI in patients presenting acutely with otherwise unexplainable neurological symptoms may facilitate diagnosis and decrease morbidity. ■

Responsabilidades Éticas

Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

Fontes de Financiamento: Não existiram fontes externas de financiamento para a realização deste artigo.

Confidencialidade dos Dados: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.

Consentimento: Consentimento do doente para publicação obtido.

Proveniência e Revisão por Pares: Não comissionado; revisão externa por pares.

Ethical Disclosures

Conflicts of interest: The authors have no conflicts of interest to declare.

Financing Support: This work has not received any contribution, grant or scholarship

Confidentiality of Data: The authors declare that they have followed the protocols of their work center on the publication of data from patients.

Patient Consent: Consent for publication was obtained.

Provenance and Peer Review: Not commissioned; externally peer reviewed.

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Received / Recebido: 08/10/2020
Accepted / Aceite: 03/12/2020

Publicado / Published: 18 de junho de 2021

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