https://revistas.udelar.edu.uy/OJS/index.php/anfamed/index
ISSN-e: 2301-1254

DOI: 10.25184/anfamed2025v12n1a6
An Facultad Med (Univ Repúb Urug). 2025; 12(1): e702

Hyperdense Basilar Artery Sign in a Young Cocaine User: A Rare Presentation of Stroke

Sinal da artéria basilar hiperdensa em um jovem usuário de cocaína: uma apresentação rara de acidente vascular cerebral

Signo hiperdenso de la arteria basilar en un joven consumidor de cocaína: una rara manifestación de accidente cerebrovascular

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RESUMO

O uso de cocaína é um fator bem documentado para acidentes vasculares cerebrais (AVC) isquêmicos e hemorrágicos, embora a trombose da artéria basilar causada por cocaína seja rara, com poucos casos descritos na literatura. Como uma droga ilícita amplamente utilizada, a cocaína pode desencadear trombose aguda da artéria basilar, resultando em desfechos neurológicos graves. Os mecanismos envolvidos incluem vasoespasmo cerebral, agregação plaquetária, trombose, vasculite cerebral e tromboembolismo devido a cardiomiopatia ou arritmias induzidas pela droga. Relatamos o caso de um homem de 30 anos com convulsões recorrentes após o uso de cocaína.

Palavras-chave: AVC Isquêmico; Tomografia Computadorizada por Raios X; Cocaína

ABSTRACT

Cocaine use is a well-documented factor in both ischemic and hemorrhagic stroke; however, basilar artery thrombosis due to cocaine use is rare, with few cases reported in the literature. As a widely used illicit drug, cocaine can trigger acute basilar artery thrombosis, leading to severe neurological outcomes. The mechanisms involved include cerebral vasospasm, platelet aggregation, thrombosis, cerebral vasculitis, and thromboembolism resulting from cardiomyopathy or drug-induced arrhythmias. We report the case of a 30-year-old man with recurrent seizures following cocaine use.

Key words: Ischemic Stroke; Tomography, X-Ray Computed; Cocaine

RESUMEN

El uso de cocaína es un factor bien documentado en los accidentes cerebrovasculares (ACV) isquémicos y hemorrágicos; sin embargo, la trombosis de la arteria basilar debido al uso de cocaína es rara, con pocos casos descritos en la literatura. Como droga ilícita ampliamente utilizada, la cocaína puede desencadenar una trombosis aguda de la arteria basilar, resultando en graves consecuencias neurológicas. Los mecanismos involucrados incluyen vasoespasmo cerebral, agregación plaquetaria, trombosis, vasculitis cerebral y tromboembolismo debido a cardiomiopatía o arritmias inducidas por la droga. Presentamos el caso de un hombre de 30 años con convulsiones recurrentes después del uso de cocaína.

Palabras clave: Accidente Cerebrovascular Isquémico; Tomografía Computarizada por Rayos X; Cocaína

Fecha de recibido: 11/11/2024
Fecha de aceptado: 27/01/2025

DOI: 10.25184/anfamed2025v12n1a6

A 30-year-old man had a seizure 30 minutes ago. He developed decreased consciousness associated with recurrent seizures, coarse limb tremors, and fever unresponsive to antipyretics. He is a cocaine user and had been abusing it before the seizures. Axial cranial computed tomography (CT) scan shows a hyperdense artery sign in the basilar artery (**Figure 1A**), and sagittal CT angiography (CTA) detects a filling defect in the basilar artery due to contrast, diagnosing basilar artery thrombosis (**Figures 1B-D**). After imaging, he progressed to loss of oculocephalic reflex, corneal reflex, ciliospinal reflex, and no motor response to painful stimuli. One hour later, in the intensive care unit (ICU), he presented decorticate posturing followed by decerebrate posturing. One day later, the patient died in the ICU.

The hyperdense artery sign is indicative of thrombosis[1]. This sign in the basilar artery is rare and has a poor prognosis[1]. Although ischemic stroke in young people is relatively rare, cocaine use is a well-established risk factor and involves multiple interconnected mechanisms[2]. Cocaine use can elevate blood pressure, triggering hypertensive crises and increasing the risk of cerebral hemorrhage[3]. It also induces vasospasm, which can cause ischemia and subsequent hemorrhage when relieved[3]. Additionally, cocaine disrupts vascular endothelium and alters blood coagulation, making vessels prone to rupture[3]. These mechanisms elevate the risks of both hemorrhagic and ischemic strokes, with platelet aggregation further contributing to ischemic events[4].

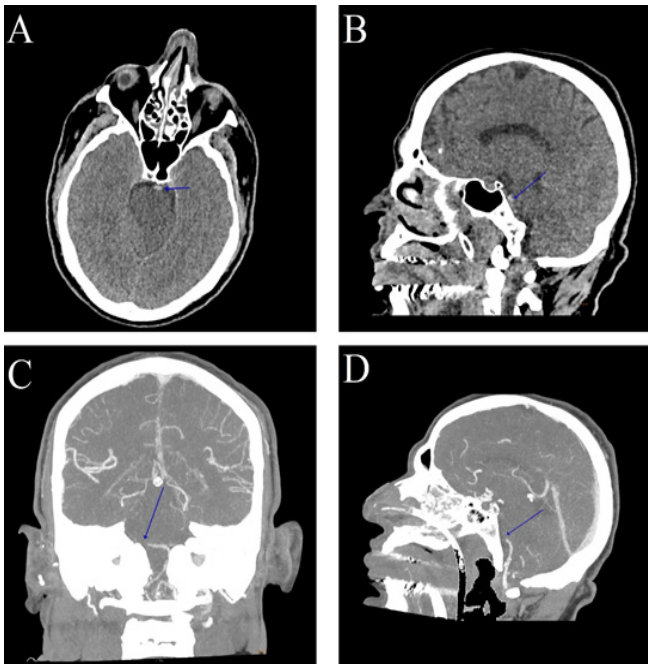

Figure 1. Non-contrast cranial CT in axial view (A) and sagittal view (B) showing the hyperdense artery sign in the basilar artery—basilar artery thrombosis (blue arrow). (B) Cranial CT angiography in MIP (maximum intensity projection) in coronal view (C) and sagittal view (D) showing a filling defect in the basilar artery due to contrast, characterizing basilar artery thrombosis (blue arrow).

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Conflict of Interest: The authors declare that they have no conflict of interest.
Funding: There´s no funding.
This article does not contain any studies with animals performed by any of the authors.
Author contribution note:
Bruno Fernandes Barros Brehme de Abreu: conceptualization (same), data curation (same), formal analysis (same), investigation (same), methodology (same), project management (same), validation (same), visualization (same), original writing-draft (same), writing-revision and editing (same);
Natã Nascimento de Jesus Graça: conceptualization (same), data curation (same), formal analysis (same), investigation (same), methodology (same), project management (same), validation (same), visualization (same), original writing-draft (same), writing-revision and editing (same);
Mayara Oliveira da Silva: conceptualization (same), data curation (same), formal analysis (same), investigation (same), methodology (same), project management (same), validation (same), visualization (same), original writing-draft (same), writing-revision and editing (same);
Felipe Nunes Figueiras: conceptualization (same), data curation (same), formal analysis (same), investigation (same), methodology (same), project management (same), validation (same), visualization (same), original writing-draft (same), writing-revision and editing (same);
Márcio Luís Duarte: data curation (same), formal analysis (same), funding acquisition (same), research (same), methodology (same), validation (same), visualization (same), original draft writing (same), writing - proofreading and editing (equal).
Declaration of data availability: All data relating to the case can be found in the article.