

Unraveling the Complexity of Cocaine-Induced Vasculitis: A Radiological Case Report in a 58-Year-Old Woman with Polysubstance Use Disorder

Daniel Eid, Ahmed-Zayn Mohamed, M.D., Katie Bailey, M.D.
The University of South Florida Morsani College of Medicine

Background:

Cocaine use has been prevalent in the United States for decades, with widespread consensus on its adverse effects on health [1]. It is theorized that levamisole is a contributing culprit in the disease process of cocaine-induced vasculitis [2]. Levamisole was originally marketed as an anti-helminthic agent but was also discovered to have immunomodulating effects, leading to its use in the treatment of cancerous and inflammatory disease up until its withdrawal from the US market in 1999 for reports of neutropenia [2]. In a 2010 study exploring ANCA-positive vasculitis and its relationship to levamisole-contaminated cocaine, it was determined there was a relation between the two, further suggesting levamisole as a prime suspect in the disease process of cocaine-induced vasculitis [3].

This study is prompted by the rarity of cocaine-induced vasculitis and the importance of familiarity with specific imaging findings indicative of this condition. The theorized mechanism of action involves antineutrophilic cytoplasmic antibody (ANCA) associated autoimmune mediated inflammation, attributed to the levamisole component often contaminating street formulations of cocaine. Additionally, the direct vasoconstrictive properties of cocaine further exacerbate its vascular manifestations.

Clinical Course:

A 58-year-old woman with a past medical history of polysubstance abuse presented to an emergency department at our institution for severe headache one week following cocaine use. On admission, MRA showed focal narrowing and dilation of the right M1 segment of the middle cerebral artery (MCA) [Figure 1a]. Additionally, MRA depicted narrowing and dilation of the left P2 segment of the posterior cerebral artery (PCA) with more distal occlusion [Figure 1b]. On examination of the anterior cerebral artery (ACA), focal narrowing and dilation of the A2 and A3 segments were also discovered [Figure 1c]. No other arterial or venous abnormality was found via MRA at the time of admission.

The patient underwent CTA of the head six weeks later, which revealed resolved right M1 dilation with some persistent narrowing [Figure 2a], resolved A2 and A3 narrowing and dilation [Figure 2b], and persistent P2 occlusion [Figure 2c]. No new arterial or venous abnormalities were identified.

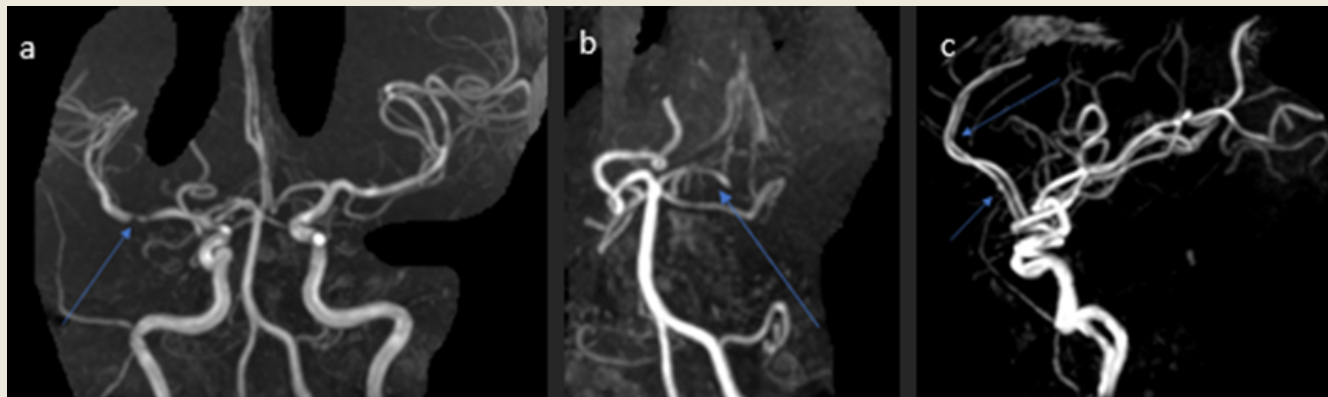


Figure 1: MRA of the head at time of presentation. (a) Coronal reconstruction depicting focal narrowing and dilation of the right M1 segment. (b) Oblique reconstruction showing PCA dilation and narrowing. (c) Sagittal reconstruction demonstrating ACA focal narrowing and dilation.

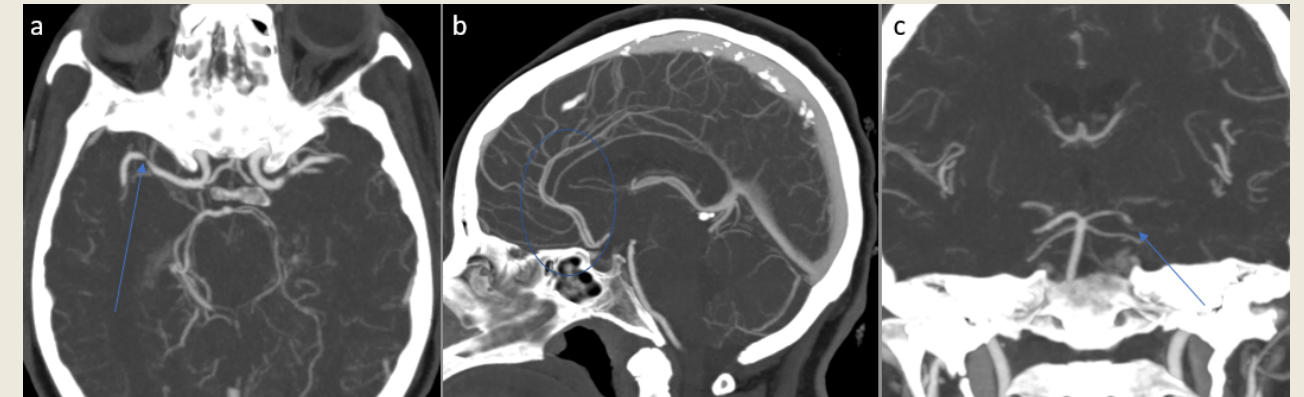


Figure 2: Six-week follow-up CTA of the head. (a) Axial view showing some persistent narrowing of the right M1 segment, but with interval resolution of the dilation originally identified. (b) Sagittal view demonstrating no residual A2 or A3 narrowing or dilation. (c) Coronal view revealing persistent involvement of the left PCA.

Discussion:

Given the above imaging findings, it is important to consider various imaging techniques in the setting of cocaine-induced vasculitis. Because significant changes in major vascular structures were present in the patient at presentation and after a six-week period, imaging becomes a highly important modality of monitoring patients with cocaine-induced vasculitis over the course of their disease. In this case specifically, the patient had improvement in MCA pathology, resolution of ACA pathology, and persistent PCA pathology [Figure 2].

Conclusion:

Due to limited research surrounding cocaine-induced vasculitis, along with the rare nature of the disease, an attempt is made to explore imaging as a modality of recognizing the disease earlier while also further monitoring vascular changes in patients. In the present case, the patient experienced varied improvement in, resolution of, as well as persistence of imaging-apparent pathology, with difficulty in identifying a pattern or course for the disease or its treatment. More study with further follow-up imaging is warranted to understand the extent of cocaine-induced vasculitis and impacts of treatment on involved vascular structures.

References

- 1) Mustaquim D, Jones CM, Compton WM. Trends and correlates of cocaine use among adults in the United States, 2006–2019. *Addictive Behaviors*. 2021;120:106950. doi:10.1016/j.addbeh.2021.106950
- 2) Berman M, Paran D, Elkayam O. Cocaine-induced vasculitis. *Rambam Maimonides Medical Journal*. 2016;7(4). doi:10.5041/rmmj.10263
- 3) McGrath MM, Isakova T, Renneke HG, Mottola AM, Laliberte K, Niles JL. Contaminated cocaine and antineutrophil cytoplasmic Antibody-Associated disease. *Clinical Journal of the American Society of Nephrology*. 2011;6(12):2799–2805. doi:10.2215/cjn.03440411