

Acute Oculomotor Nerve Palsy Caused by Compression from an Aberrant Posterior Communicating Artery: Case Report

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Abstract

Oculomotor nerve palsy is a rare neurological impairment linked to various conditions. Aneurysms of the posterior communicating artery are the most documented. However, less commonly discussed are other vascular anomalies and their potential role in causing ONP. Here, we present a case of ONP caused by a congenital vascular aberrancy.

Keywords: Palsy; Posterior Communicating Artery; Oculomotor Nerve; Vascular; Nerve Compression

Introduction

Cranial nerve palsy, particularly involving the third cranial nerve, often stems from its intricate anatomical location as it exits the brainstem, nestled between the uncus and the tentorium.

Acute presentation of third cranial nerve palsy commonly signals the presence of an unstable aneurysm, with the posterior communicating artery (PCOM) being a frequent culprit. These aneurysms, primarily found in the basal cistern, often manifest as oculomotor nerve palsy (ONP), either through direct compression or rupture [1]. Recognizing ONP as a potential indicator of aneurysmal instability is crucial, prompting immediate investigation to rule out this diagnosis [2].

Despite the minority of ONP cases being attributable to aneurysm, thorough evaluation is essential. Various clinical features and examination findings aid in differentiating aneurysmal ONP, with imaging modalities like magnetic resonance angiography (MRA), computed tomography angiography (CTA), and digital subtraction angiography (DSA) serving as gold standards.

However, diagnostic challenges persist, as exemplified by a case where ONP initially suspected to be due to PCOM aneurysm was revealed to stem from a tortuous PCOM.

Case History

A 76-year-old man, known for diabetes under treatment with no other medical or surgical history, presented with a gradually worsening left-sided incomplete ptosis over the course of three weeks.

Methods

Upon examination, his pupils were equal and reactive to light and accommodation, and there were no other focal neurological deficits observed.

Outcome and follow-up

Preceding the onset of symptoms, a brain CT scan with and without contrast injection revealed a dilated appearance of the left posterior communicating artery, whose tortuous course is likely causing compression on the third nerve.

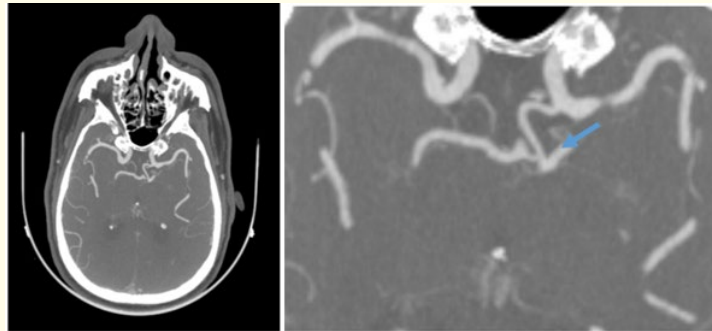


Figure 1: Brain CT scan in axial sections after contrast injection showing a tortuous appearance of the left posterior communicating artery (arrow).

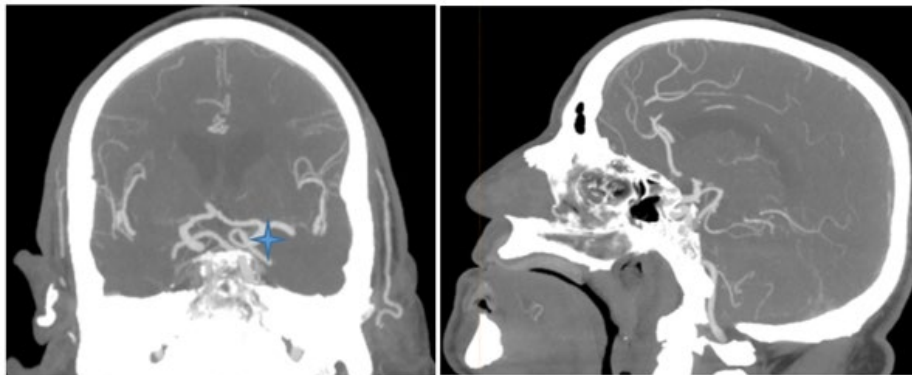


Figure 2: Brain CT scan in coronal and sagittal sections after contrast injection showing a dilated appearance of the posterior communicating artery, forming a loop-like structure (Asterisk).

Discussion

Third nerve palsies, particularly when fully or almost fully developed, result in the affected eye adopting a “down-and-out” position, primarily because the superior oblique and lateral rectus muscles maintain their function, causing the eye to depress and abduct. Consequently, substantial weakness in elevation, depression, and adduction is anticipated. Numerous third nerve palsies are considered “incomplete,” wherein not all muscles receiving innervation are affected, or the weakness in each affected muscle is less than total [3].

Common causes of oculomotor nerve palsy (ONP) include trauma, neoplasms, stroke, post-surgical inflammation, and microvascular damage from chronic disease, with microvascular damage being the most prevalent [4].

To provide context, among the 1,130 retrospective cases of third nerve palsy documented by the Mayo Clinic from the 1950s to the 1980s, encompassing patients of all age groups, 270 cases were attributed to unknown causes, 225 cases to microvascular ischemia, 179 cases to aneurysms, 166 cases to head trauma, 141 cases to neoplasms, and 149 cases to other etiologies. It's important to acknowledge that these cases were not assessed using modern neuroimaging techniques, which means that small compressive lesions might have gone undetected [5].

While history and physical examination are crucial, supporting imaging remains the most reliable diagnostic tool.

This case highlights the potential limitations of neurovascular imaging techniques, emphasizing the importance of considering aberrant anatomy in the differential diagnosis, which may require further evaluation with high-resolution MRI.

In the literature, a case has been reported of a patient presenting with third nerve palsy, in whom imaging diagnosis initially suggested a posterior communicating artery aneurysm, for which she underwent surgery. However, intraoperative findings revealed the absence of an aneurysm, with the presence of an aberrant course of the posterior communicating artery, causing compression on the third nerve [6].

Palsy of the third cranial nerve due to anatomic aberrancy is exceedingly rare, with variation of the posterior cerebral artery being more commonly documented. As far as we know, there have been eight documented cases of aberrant posterior cerebral artery (PCA) causing oculomotor nerve palsy (ONP) [7], but only one instance reported in the literature of aberrant posterior communicating artery (PCOM) causing ONP [6].

Assessing third cranial nerve palsies through radiological means remains exceptionally challenging and should be performed by a neuroradiologist with extensive experience and fellowship training.

Conclusion

Timely identification and assessment of third cranial nerve palsy are crucial to exclude the possibility of a posterior communicating artery (PCOM) aneurysm. While uncommon, aberrant vasculature involving the posterior cerebral artery (PCA) or PCOM can lead to oculomotor nerve palsy (ONP) and should therefore be considered in the differential diagnosis for these patients, particularly when diagnostic imaging fails to provide conclusive evidence.

Patient Consent Statement

In accordance with ethical guidelines and institutional regulations, informed consent was obtained from all participants included in this study. Each participant was provided with detailed information regarding the nature, purpose, and potential implications of the research. They were informed that their participation was voluntary and that they could withdraw at any time without any consequences to their treatment or relationship with the healthcare provider.

For the purpose of this report, patients were assured that their privacy and confidentiality would be maintained at all times. No identifiable personal information has been disclosed, and all data have been anonymized. The study protocol was reviewed and approved by the relevant institutional review board (IRB)/ethics committee.

By signing the consent form, participants acknowledged their understanding and agreement to participate in the study and to have their anonymized data used for research and publication.

Author Contributions

All authors contributed significantly to this work. A. Sekkat conducted the experiments, collected, and analyzed the data. Y. Lamrani conceived and designed the study, provided overall supervision, and drafted the manuscript. M. Maaroufi critically revised the manuscript. All authors reviewed and approved the final manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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