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Cranial Nerve III Palsy and Nothnagel Syndrome in a Patient with Vascular Risk Factors: A Case Report

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INTRODUCTION

Cranial nerve III (CN III) palsy with pupillary involvement often is attributed to aneurysmal compression, making it a critical diagnosis to consider. However, midbrain syndromes such as Nothnagel syndrome demonstrate that ischemic infarction can produce a similar clinical presentation.[1] Careful bedside examination remains essential for accurate lesion localization and to avoid misdiagnosis. This case of CN III palsy accompanied by contralateral ataxia highlights the importance of recognizing Nothnagel syndrome as a rare but instructive manifestation of midbrain pathology.

CASE REPORT

An 80-year-old man presented with acute diplopia and imbalance. Physical examination revealed a dilated, minimally reactive right pupil, limited extraocular movements (EOMs), ptosis secondary to levator palpebrae involvement, and left-sided ataxia. Magnetic resonance imaging (MRI) of the brain without contrast demonstrated subacute infarcts involving the medial midbrain and superior cerebellar peduncle (Figure 1). Following admission, dual antiplatelet therapy was initiated, and blood pressure was closely monitored. After stabilization, he was discharged to an inpatient rehabilitation facility with the goal of returning to his baseline level of function. Prior to admission, he reported mild balance difficulties and required moderate assistance when navigating stairs and uneven surfaces. During hospitalization, his pupillary reactivity and diplopia improved progressively. By discharge on hospital day 11, he required only supervision-level assistance and demonstrated notable improvement in mobility, pupillary function, and extraocular movements, which were approaching baseline.

DISCUSSION

Nothnagel syndrome is a rare midbrain syndrome that often is underdiagnosed because of its complex clinical presentation. It results from lesions involving the CN III fascicle and superior cerebellar peduncle.[1] This case highlights the value of integrating physical examination findings with neuroimaging to distinguish between compressive

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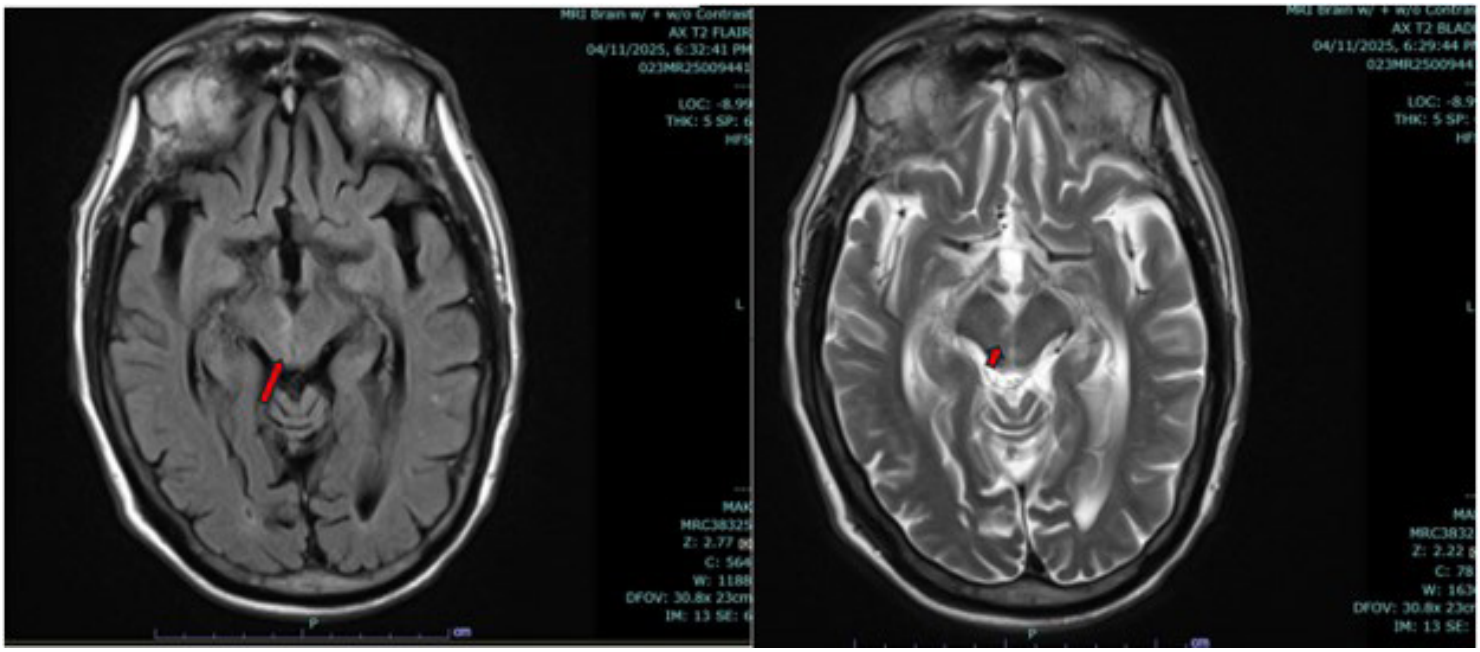

Figure 1. A small focus of T2/FLAIR hyperintensity in the right medial midbrain in the territory near the cerebral peduncle indicated with the red arrows consistent with a lesion relevant to Nothnagel syndrome.

and microvascular causes of CN III palsy.

Although non-pupil-sparing CN III palsy classically raises concern for aneurysmal compression,[2,3] this case demonstrates that microvascular ischemia, particularly in patients with vascular risk factors such as hypertension and hyperlipidemia, can produce a similar presentation. Initial concern for aneurysmal compression was warranted given the patient's pupillary involvement. However, progressive improvement in extraocular movement function, levator palpebrae strength, and resolution of mydriasis during rehabilitation supported a microvascular ischemic etiology.[3] This conclusion was further supported by the patient's vascular risk profile, which included hyperlipidemia, peripheral vascular disease, and tobacco use.

CONCLUSIONS

In elderly patients with vascular risk factors, CN III palsy with pupillary involvement does not necessarily indicate an aneurysm. This case underscores the importance of neuroimaging, clinical progression, and risk-factor assessment in guiding the diagnosis and management of rare midbrain syndromes such as Nothnagel syndrome.

ARTICLE INFORMATION

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