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# Optic-Pyramidal Syndrome and Ophthalmoplegia Due to Internal Carotid Artery Occlusion: A Case Report

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**Abstract** **Case Report**

This case report highlights the neuro-ophthalmological complications, associated with internal carotid artery occlusion, particularly optic-pyramidal syndrome. We present the case of a 73-year-old female patient with a history of hypertension, admitted for right-sided hemiparesis and decreased visual acuity in the left eye, accompanied by ptosis. Imaging revealed a left fronto temporal ischemic stroke with occlusion of the internal carotid artery. We draw practitioners' attention to the potential occurrence of ophthalmoplegia in cases of optic-pyramidal syndrome. A carotid occlusion with inadequate collateral circulation from the vertebrobasilar system could explain the symptomatology, which may be transient or, in rare cases, permanent.

**Keywords**: Ophthalmoplegia, Ischemic stroke, Carotid occlusion, Optic-pyramidal syndrome.

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## INTRODUCTION

Optic-pyramidal syndrome is a rare and classic manifestation associated with internal carotid artery involvement, often caused by stenosis. This syndrome is characterized by transient reduction in visual acuity on the same side as the carotid lesion, accompanied by a transient motor deficit on the opposite side. The occurrence of ptosis within this syndrome is uncommon. It is also known as Espildora Luque syndrome, first described in 1934 by ophthalmologist Carlos Espildora Luque [1]. In his study, he reported three cases exhibiting transient monocular blindness combined with contralateral hemiplegia, confirmed through cranial arteriography [2]. This clinical picture is thought to result from an embolism in the ophthalmic artery, inducing a reflex spasm in the ipsilateral middle cerebral artery. This rare syndrome illustrates the complexity of interactions between the cerebral and ophthalmic vascular networks. Here, we present the case of a patient with optic-pyramidal syndrome accompanied by left-sided ophthalmoplegia on the same side, due to left internal carotid artery occlusion.

## CASE REPORT

We present the case of a 73-year-old female patient with a 15-year history of hypertension managed with a calcium channel blocker, admitted six hours after the onset of symptoms due to sudden-onset right-sided hemiparesis, reduced visual acuity, and ptosis of the left eye on the contralateral side, with no impairment of the pupillary light reflex.

Ophthalmological examination of the right eye revealed severely reduced visual acuity, limited to light perception at 1/10. The anterior segment and intraocular pressure were normal. Funduscopic examination identified a central retinal vein occlusion. Other cranial nerves, including the sensory component of the trigeminal nerve, were unaffected. The remainder of the neurological examination was unremarkable aside from the initial symptoms. The cardiovascular examination revealed no cardiac murmur, although a left carotid bruit was detected, and peripheral pulses were well palpated and symmetrical. The general examination showed no additional abnormalities.

Magnetic resonance imaging (MRI) (Fig 1) and cerebral MR angiography (Fig 2) demonstrated a left frontotemporal ischemic stroke associated with acute occlusion of the left internal carotid artery extending to the ipsilateral middle cerebral artery. CT angiography of the supra-aortic trunks and the circle of Willis confirmed similar findings, including significant atherosclerosis of the supra-aortic trunks. Laboratory analyses were

normal, with no detected abnormalities, anti-DNA, APL, and ANA autoantibodies were negative, and the inflammatory markers were within normal limits.

The patient was scheduled for regular follow-up with a planned endarterectomy to address the internal carotid artery occlusion. Ophthalmological monitoring was prioritized, although no recovery in visual acuity was observed.

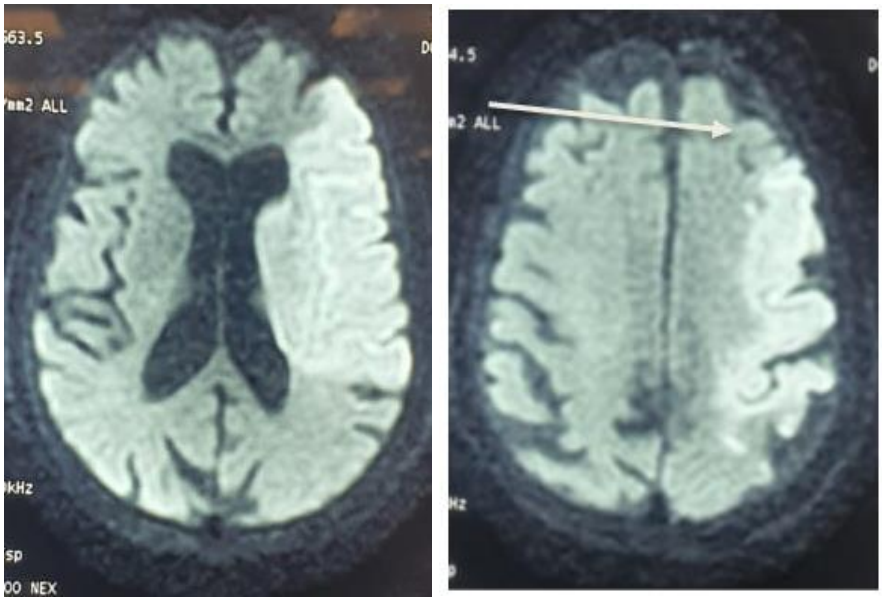

**Figure 1: Brain MRI showing hyperintense areas in the left insular and temporal lobes on diffusion-weighted imaging and ADC restriction, with corresponding changes on FLAIR**

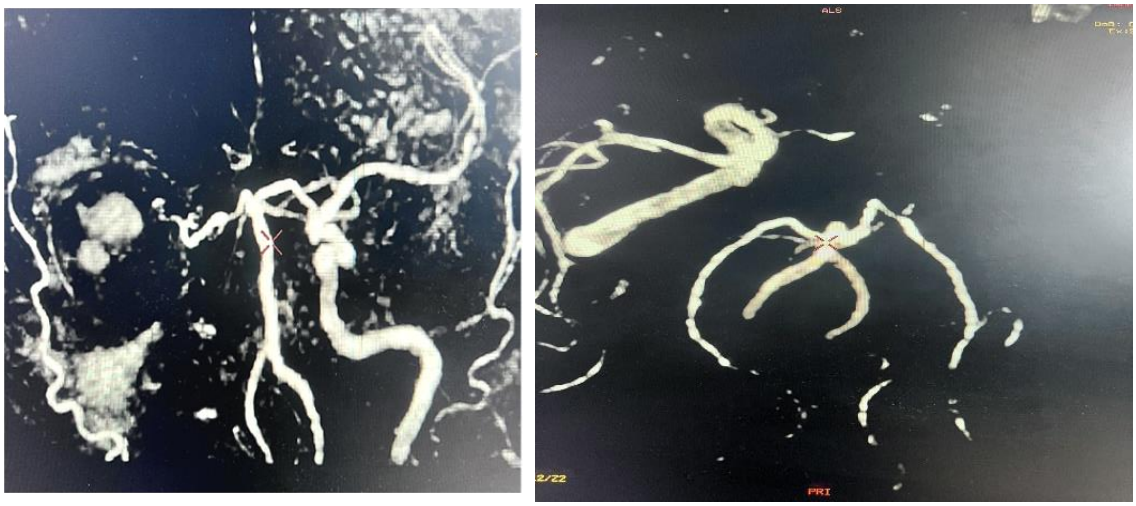

**Figure 2: Brain MRI with 3D TOF sequence: occlusion of the left carotid artery extending to the ipsilateral Sylvian artery, patent basilar trunk, patent vertebral arteries with parietal calcifications in the intracranial space narrowing the lumen in places**

## DISCUSSION

As described in the literature, stenosis of the internal carotid artery leads to a reduction in blood perfusion to the ophthalmic artery, causing ischemic ophthalmopathy that affects the extraocular muscles, retina, and optic nerve [3]. The association of the optico-pyramidal syndrome with ophthalmoplegia suggests vertebrobasilar involvement in conjunction with carotid artery pathology. Ophthalmic involvement can be the first manifestation of occlusive disease, presenting with a varied clinical picture, often characterized by transient monocular blindness [4]. This is frequently due to platelet emboli or cholesterol deposits originating from a recent thrombus or an ulcerated plaque in the internal carotid artery. The occurrence of this symptom serves as a marker for atherosclerotic disease and represents an equivalent of transient ischemic attack, carrying neurological and ocular risks [5, 6]. Its appearance warrants exploration of carotid circulation, as the risk of early stroke recurrence is significantly higher in patients

with carotid stenosis compared to those with cardioembolic stroke [7].

In the presence of such a clinical picture, carotid pathology should be suspected, and a vascular assessment must be conducted to confirm and evaluate the severity of the involvement. This evaluation relies on Doppler ultrasound of the neck vessels, CT angiography, and particularly MRI angiography with two-dimensional or three-dimensional reconstruction. Electrophysiological examinations of the eye, such as electroretinography and visual evoked potentials, may also reveal abnormalities, especially in subclinical stages where angiography appears normal [8].

Management aimed at reducing the risk of stroke in patients with carotid stenosis includes the administration of antiplatelet agents, control of cardiovascular risk factors (hypertension, diabetes, hyperlipidemia), and lifestyle modifications such as smoking cessation. Surgical interventions for severe symptomatic carotid stenosis (with a history of transient ischemic attack or ipsilateral stroke) or asymptomatic stenosis (often detected through vascular imaging or during the auscultation of a carotid bruit) include carotid endarterectomy and angioplasty with stenting. There remain uncertainties regarding the indications and comparative effectiveness of these two techniques. Although several randomized clinical trials conducted in Europe and North America have explored this topic, the results remain contradictory. Furthermore, carotid surgery generally does not improve long-term visual prognosis, particularly when iris rubiosis is present and initial visual acuity is low, as reported by the study by Mizener *et al*., [9].

Overall, the optico-pyramidal syndrome, when associated with ophthalmoplegia, suggests carotid involvement combined with vertebrobasilar impairment. In our case, the CT angiography of the supraaortic trunks and the circle of Willis ruled out vertebral artery involvement as it was of normal caliber and patent, as was the basilar trunk. Carotid origin was established as the cause. The ptosis secondary to oculomotor involvement (left third cranial nerve involvement) could be explained by the failure of the compensatory system and therefore an insufficiency of the collaterals of the vertebrobasilar system, exacerbated by hypertensive microangiopathy. The pathophysiology is likely related to ischemia and paralysis of the oculomotor nerves, which can be either transient or, more rarely, permanent as in our case.

## CONCLUSION

This case illustrates the impact of internal carotid artery occlusion on visual and neurological functions, presenting with an optico-pyramidal syndrome and ophthalmoplegia. This observation underscores the importance of rapid diagnosis and a multidisciplinary approach, as well as the management of risk factors to improve prognosis. Further research is needed to better understand the connection between optic and pyramidal symptoms, in order to develop more targeted therapeutic protocols.

**Consent:** Written informed consent was obtained from the patient's family for the publication of anonymized information in this case report.

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**Author Contributions**

Mounir Lekhlit and Nada Benabdelouahab contributed as principal authors, involved in the conception, design, and writing of the case report.

Sabrine Kamal, Chourouk Regragui, and Jehanne Aasfara assisted with data collection and manuscript revisions.

Saad Zidouh and Lahcen Belyamani, as supervising professors, provided guidance and oversight throughout the study.

All authors have read and approved the final manuscript.

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**Data availability:** Data supporting the findings of this study are openly accessible within the article.

**Ethical approval:** Ethical approval of case report in not needed in accordance with the local ethical guidelines.

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