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# Intracranial Aneurysm Revealing a Primary Open-Angle Glaucoma in Progression: A Case Report from the Albert ROYER National Children's Hospital

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**Abstract** **Case Report**

Glaucoma is the world's second leading cause of blindness. Steadily increasing, the population of those affected is estimated to reach 76 million; its aggravating factors are well known. Being able to be isolated or associated with other pathologies, the discovery of an intracranial aneurysm would not be impossible.

**Keywords:** Glaucoma, intracranial anevrysm.



## INTRODUCTION

Glaucoma is the second leading cause of blindness worldwide [1]. A 2018 meta-analysis estimated the global patient population in 2020 at around 76 million. Africa is particularly affected, with a relative risk of 2.8 compared with Europe [1]. It is not uncommon to diagnose other pathologies associated with this optic neuropathy as aggravating or non-aggravating factors [1].

An intracranial aneurysm is always the consequence of a structural change in the arterial wall responsible for this loss of parallelism [2]. The origin of this parietal lesion is very varied and remains hypothetical. The prevalence of intracranial aneurysms varies depending on whether the study was prospective or retrospective, and on the population studied. Depending on the series, it varies from 0.8% to 4.6% in the general population, with a predominance of women (sex ratio 2.2-2.4) [3]. The risk factors for intracranial aneurysms are age, female gender, active smoking and high blood pressure [3]. In 80% of cases, the aneurysm is single and less than 12 mm in size, with a saccular shape in 97% of cases. Topographically, it is located on the anterior part of the polygon of Willis in 95% of cases [4]. Whatever the case, the location, shape, evolution and mode of discovery of an intracranial aneurysm depend on the aetiology. Aneurysms tend to increase in volume under the influence of haemodynamic factors, leading to a weakening of the aneurysm wall. This is why rupture of the aneurysm is the most frequent mode of discovery [4]. Intracranial aneurysms are a major concern for neurosurgeons, intensive care units and neuroradiologists because of the diagnostic, therapeutic and prognostic problems involved [5]. To illustrate this point, we report a case of intracranial aneurysm in glaucoma.

## OBSERVATION

This is a 50-year-old male patient from Dakar who was brought in for a 3-year history of progressively worsening visual acuity in both eyes, predominantly on the right. He had consulted a local health facility where he was diagnosed with primary open-angle glaucoma (POAG). The patient was put on alpha adrenergic medication (Brimonidine eye drops: one drop x 2 /d in both eyes).

Six months prior to the current consultation, he presented with a sudden onset of left hemiplegia associated with a documented ischaemic stroke. About three weeks ago, a left unilateral ptosis preceded by a violent headache occurred. The symptoms worsened with the onset of diplopia and hemiplegia contralateral to

the ptosis, prompting specialist ophthalmology and neurology consultations respectively.

It should be noted that the patient had been taking Amlodipine 10 mg tablets (1 tablet x 2/d) for approximately 5 years. There were no reports of treatment discontinuation.

The clinical examination revealed the following:

- Bilateral blindness;
- Total paralysis of the right common oculomotor nerve (III) with mild ptosis, divergent strabismus and photomotor areflexia; partial paralysis of the left of the left common, oculomotor nerve (III) with divergent strabismus, severe ptosis and photomotor hyporeflexia;
- Ocular hypertonia of 38 mmHg in the right eye and 46 mmHg in the left eye
- Optic atrophy in the right eye and an excavation of 0.9 in the left eye;
- A right pyramidal syndrome of recent onset, with global and proportional hemiplegia proportional hemiplegia and osteotendinous areflexia;
- A left pyramidal syndrome of sequellar appearance, with global and proportional hemiparesis and osteotendinous hyperreflexia.

As a matter of urgency, we prescribed topical dual therapy with a prostaglandin beta-blocker analogue, acetazolamide 250 mg tablets (twice daily) and a potassium sparing agent 600 mg Lp tablets.

The possibility of a cerebral aneurysm was raised, and the patient was referred to neurology for clinical, morphological and biological investigations (cerebral MRI angiography, ultrasound of the supra-aortic trunks, cardiac ultrasound, lipid profile, blood cell count, creatininaemia, uraemia, transaminases).

MRI confirmed the presence of a partially thrombosed saccular aneurysm of the left posterior communicating artery, 12 x 10 mm long with a 2.6 x 2.7 mm neck. Moderate triventricular hydrocephalus was also detected, with no visible subarachnoid haemorrhage and no newly formed ischaemic focus (see figures below).

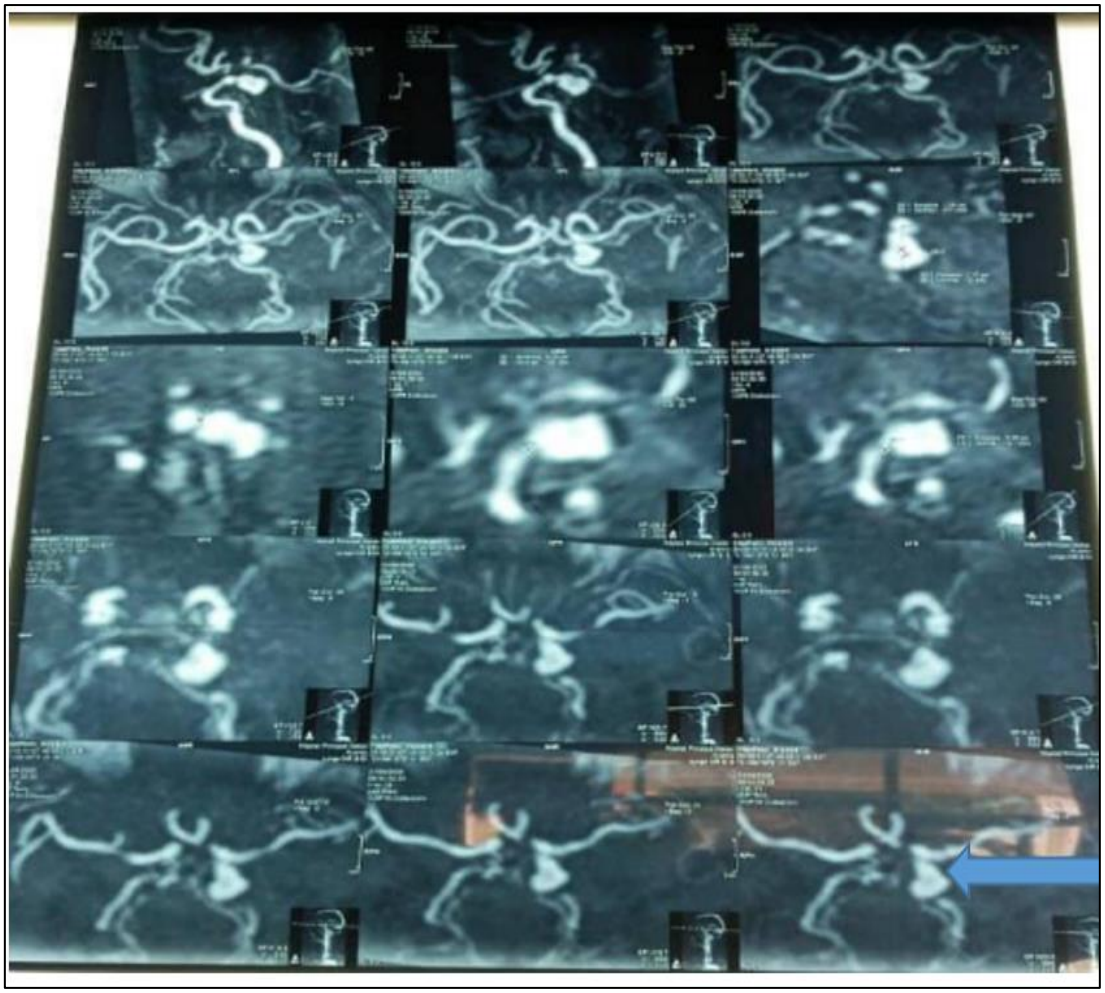

**Figure 1: MRI angiography showing a saccular aneurysm of the left posterior communicating artery**

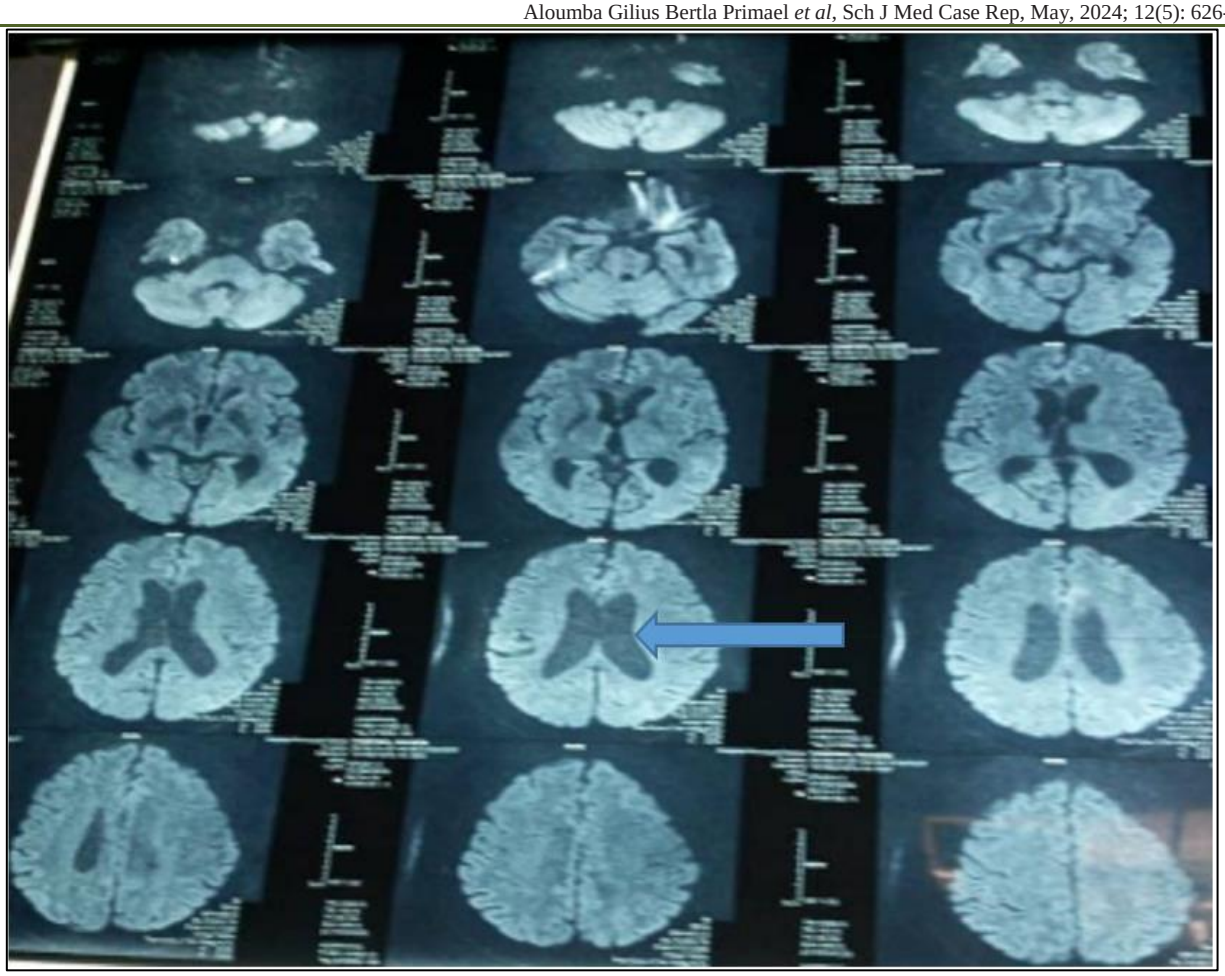

**Figure 2: Ventricular dilatation in axial section of MRI angiography**

Ultrasound of the supra-aortic trunks revealed atheromatosis of the carotid bulbs with non-stenosing calcified plaques.

LDL-cholesterol dyslipidaemia was also detected (1.96 g/l).

The indication for surgery was established, and the neurosurgeons were asked to continue the treatment. However, five days after the hypotonisers were prescribed, eye tone fell (TO: 22 mm Hg OD and 26 mm Hg OG). Topical hypotonisers were maintained; with functional prognosis counselling (visual function), the clinical aspects of oculomotor nerve ophthalmoplegia would be better assessed after surgery.

## DISCUSSION

The posterior communicating artery is a branch of the polygon of Willis. During its course, it provides communication between the middle cerebral artery at the front and the posterior cerebral artery at the back [6]. Pathophysiological processes are likely to affect this artery, making it a potential area of weakness. However, it may rupture, causing either intra-parenchymal haemorrhage or meningeal haemorrhage [7]. The fortuitous discovery of an aneurysm in this vascular territory is most often possible when the clinical symptoms are strongly suggestive of nerve III ophthalmoplegia; this was the case for our patient. Furthermore, the occurrence of headaches preceding the clinical picture is characteristic of intracranial hypertension, a sign spontaneously reported by patients [2,8-10].

Our patient presented with an alternating Weber-type syndrome associating a ptosis of the left nerve III and a contralateral pyramidal syndrome. Given its particularly long course, ptosis is thought to be the result of compression of this nerve [6].

Aetiological research allows us to distinguish between congenital causes and acquired causes incriminated in the genesis of intracranial aneurysms [6,7], including atherosclerosis. High blood pressure is one of the vascular risk factors for stroke. Both of these factors were present in our patient.

Although the pathophysiology of GPAO is not fully understood, theories:

- Mechanical, which explains papillary excavation by compression of the head of the optic nerve under the effect of ocular hypertonia,

- And ischaemic, which explains it by circulatory insufficiency at the level of the blood capillaries of the optic nerve head (by chronic papillary hypoperfusion or transient vascular spasms) are verified with the clinical presentation of our patient's clinical presentation.

Furthermore, on the basis of the ischaemic hypothesis favouring cell death phenomena leading to the destruction of visual fibres, the formation of atheromatous plaques on the carotid arteries would partly account for this ischaemia, since the central retinal and ophthalmic arteries are dependent on the internal carotid artery.

The purpose of requesting an ultrasound of the supra-aortic trunks and a blood lipid test was to identify the factors that could potentially have aggravated his glaucoma. Atherosclerosis (linked to dyslipidaemia) and hypertension were found to be aggravating factors in glaucoma.

Orbito-cerebral MRI remains the first-line examination, enabling not only a lesion diagnosis but also a topographical diagnosis to be made [2]. Considerable therapeutic advances (embolisation and clip placement) in neurosurgery have improved the vital and functional prognosis of patients.

The average age of discovery is close to 50. Hypertension prior to rupture is estimated to be present in 21% to 45% of cases [4]. Intracranial saccular aneurysms are less than or equal to 10 mm in almost 70% of cases. These anamnestic and clinical data corroborate those found in our patient.

## CONCLUSION

In short, GPAO is an optic neuropathy which may or may not be isolated. Any progression should always prompt a search for aggravating factors. The discovery of this aneurysm is an epiphenomenon which will have enabled early management with the aim of preventing further episodes of stroke.

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