

Idiopathic Intracranial Hypertension Revealed by Papillary Oedema: About Two Cases at the Mauritius Eye Clinic in Guinea

Maxime Dantouma SOVOGUI^{1,2*}, Abdoul Karim BALDE⁴, Christophe ZOUMANIGUI², Mamadou Bobo DOUKOURE³ and Kokou VONOR⁵

¹Faculty of Health Sciences and Techniques / University of Conakry, Republic of Guinea

²Bartimee Ophthalmological Clinic, Republic of Guinea

³Kankan Regional Hospital, Republic of Guinea

⁴Donka National Hospital University Hospital Center, Republic of Guinea

⁵University of Kara in Togo, Republic of Guinea

*Corresponding Author

Dr. Maxime Dantouma SOVOGUI, Assistant at the Faculty of Health Sciences and Techniques, Gamal Abdel Nasser University. BP: 2525 Conakry, Republic of Guinea.

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Summary

Objective: To describe the ophthalmological manifestations of idiopathic intracranial hypertension at the Mauritius Clinic.

Patients and Observations: We report two cases of idiopathic intracranial hypertension revealed by papillary edema, received in consultation at the Mauritius Eye Clinic. The symptomatology was dominated by visual eclipses, headaches and tinnitus. Decreased visual acuity was associated in patient No. 2. Papillary edema was found in both grade III cases and respectively. Consultations, paraclinical and I examinations in neurosurgery and infectiology were not contributory. The patients all benefited from a drug treatment based on acetazolamide 250mg. Cortancyl 20mg with a low-calorie diet have been combined with the treatment of patient No. 1. The evolution was favorable in patient No. 1 while patient No. 2 was at risk of visual loss due to the stage of papillary edema.

Conclusion: Faced with papillary edema associated with visual disorders, headaches, tinnitus and/ or not a decrease in visual acuity, it is necessary to suspect idiopathic intracranial hypertension until proven otherwise.

Keywords: Guinea, Idiopathic intracranial hypertension, Papillary edema

1. Introduction

Idiopathic intracranial hypertension (IIH) is the increase in cerebrospinal fluid (CSF) pressure in the absence of any apparent brain damage [1]. It is a pathology of low frequency in the general population with 1 to 2 cases / 100,000 inhabitants, but twenty times more common in women of childbearing age and overweight [2,3]. Much more rarely, it affects men, children and the elderly in whom the association with obesity is less common than in young women [4]. It is isolated intracranial hypertension (ICPH) with no apparent cause [5]. Its diagnosis is evoked before: papillary edema which is a fluid and / or axonal swelling of the head of the optic

nerve following a blockage of axoplasmic flow at the level of the screened lamina, headache, decreased visual acuity, absence of an expansive intracranial process by neuroimaging and normal biological analysis of CSF [6,7]. Its main complication is irreversible blindness [8,9]. The rarity of the pathology and its impact on vision are the reasons that motivated the realization of this study. We report here two cases, the first is a 50-year-old woman and the second case is a 12-year-old boy. The objective of this study was to describe the ophthalmological manifestations of idiopathic intracranial hypertension at the Mauritius Clinic in Guinea.

2. Patients and Observations

2.1 Case 1

This was a 50-year-old patient, received in our department on March 27, 2023. Visual eclipses characterized the clinical picture, myodesopsia in the right eye and blurred vision, tinnitus. The whole was associated with pulsatile headaches localized in the right retroorbital region, radiating to the neck, aggravated by eye movements, and resistant to usual analgesics. The evolution was 5 days. As a history, she had high blood pressure diagnosed and treated for 5 years with calcium channel blocker (Amlodipine 5mg). The patient is sedentary overweight with a body mass index (BMI) equal to 31.8 kg/m². No known notion of alcoholism, smoking or drug allergy was found. The ophthalmological examination was bilateral and comparative, eye-by-eye and plane by plane.

2.2 To the Right Eye

Visual acuity from afar without correction was 10/10th.

With the slit lamp: healthy appendages, transparent cornea; the quiet and deep anterior chamber; the round pupil centered with a photomotor reflex present, the IOP measured at flattening was 16 mm Hg.

Fundus examination after obtaining maximum pupillary dilation revealed stage I papillary oedema.

2.3 In the Left Eye

Visual acuity from afar without correction was 10/10th.

With the slit lamp and fundus, the clinical features found were identical to those found in the right eye described above.

A syndrome of intracranial hypertension was evoked in front of this table.

Retinography and RNFL-OCT performed confirmed bilateral stage I papillary oedema (Figures 1 and 2). The automated visual field (VF) performed showed a peripheral alteration of the VF (Figure 3). The patient was referred to the neurosurgery department where brain magnetic resonance imaging (MRI) coupled with venous angiography sequences were performed concluding that there was tortuosity of the optic nerves with a discrete enlargement of the peri-optic space, a partially empty turcic saddle and an enlargement of the subarachnoid spaces and basic tanks (Figure 4). Thus, eliminating an expansive intracranial process and a possible vascular cause.

In the infectious and tropical diseases department, she benefited from paraclinical biological examinations: complete blood count and retroviral serology, which revealed no peculiarities. Blood biochemistry showed fasting hyperglycemia of 1.38g/dl. This allowed us to eliminate an ongoing infectious process.

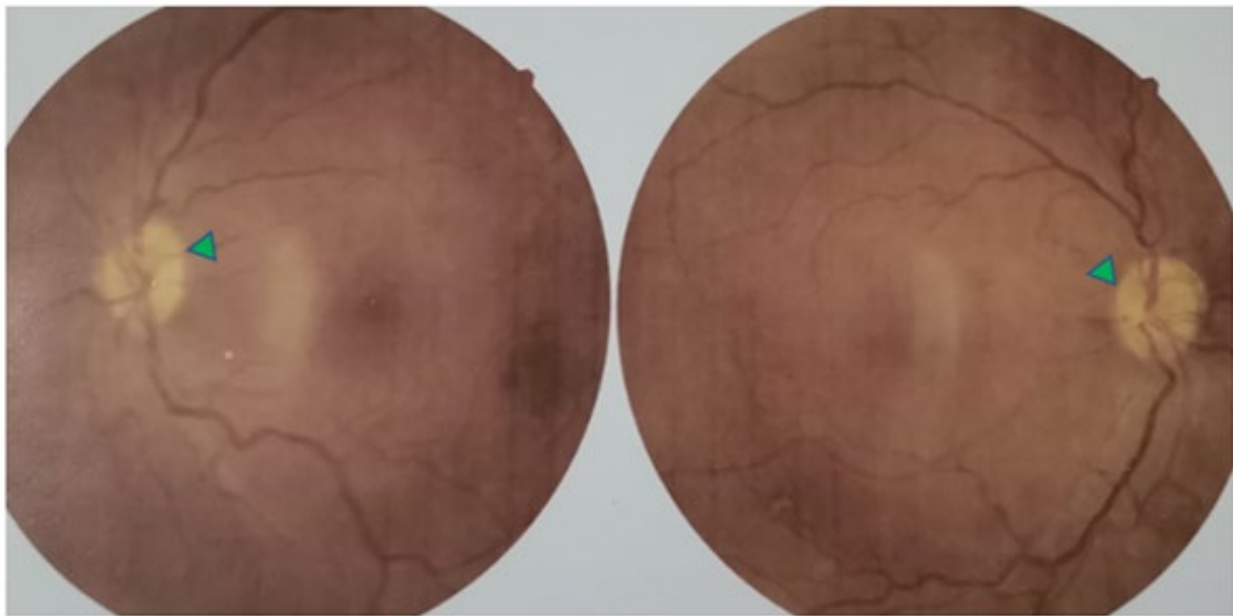


Figure 1: Photo retina right eye and left eye, highlighting stage I papillary edema (see green arrow)

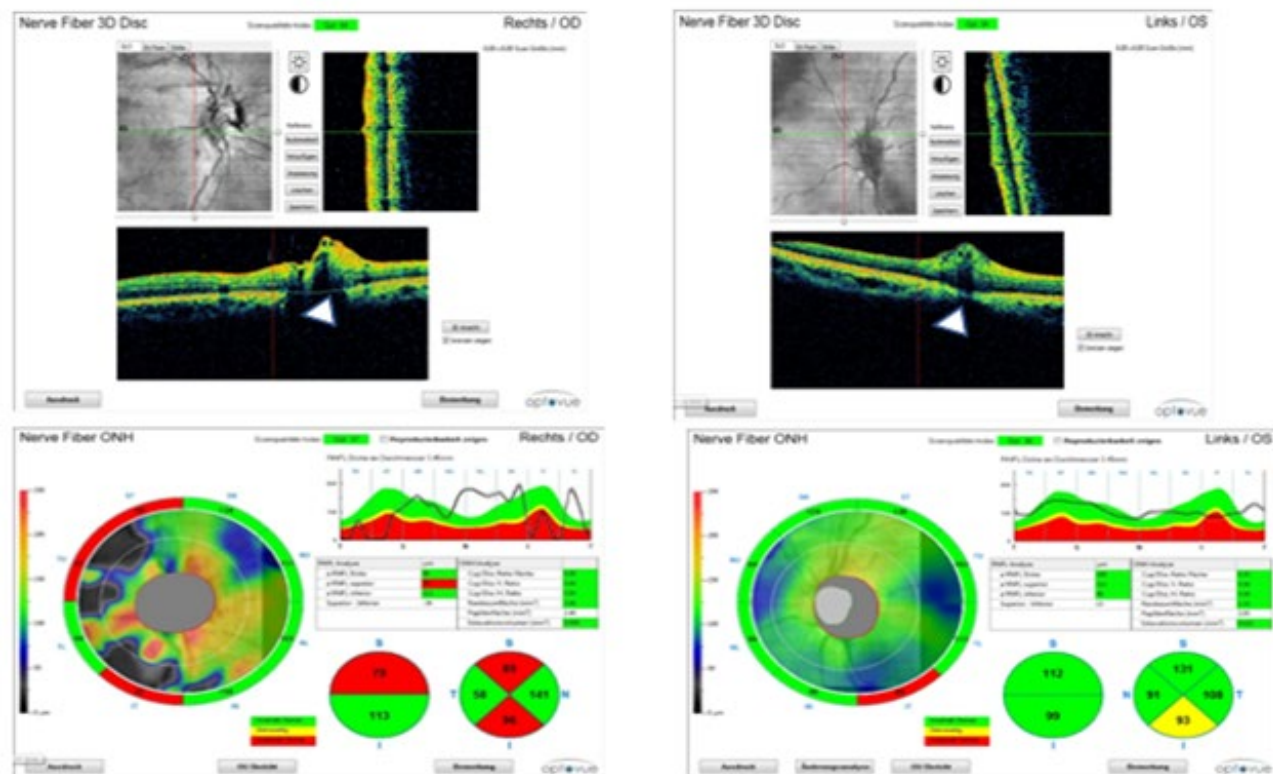


Figure 2: OCT - RNFL right and left eye, highlighting stage I papillary oedema

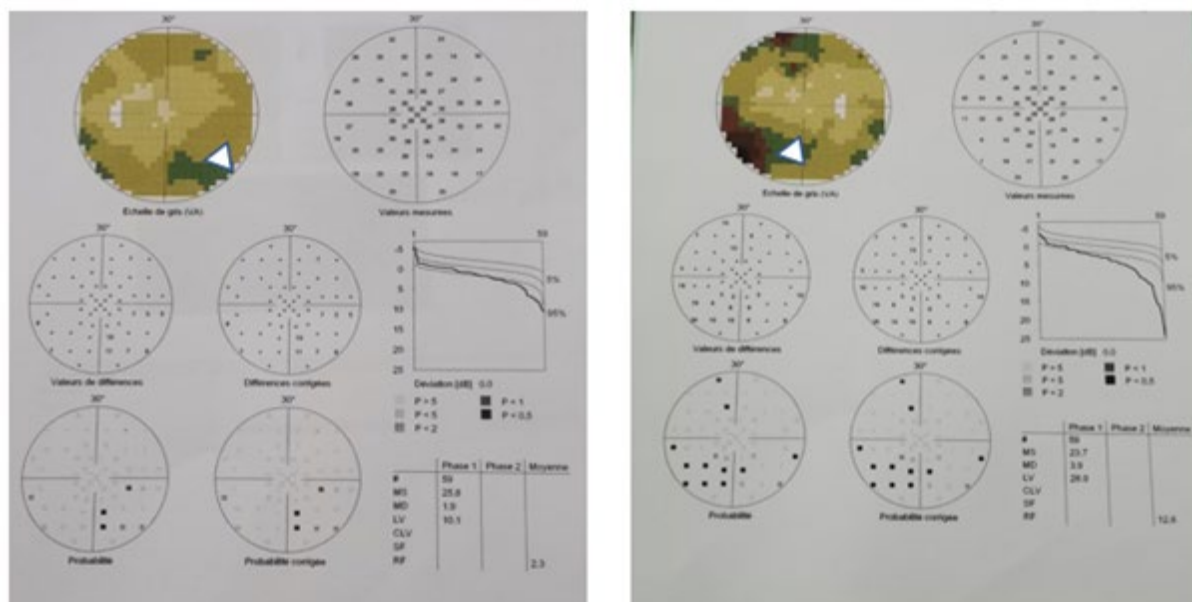


Figure 3: Visual field highlighting peripheral corruption (see white arrow)

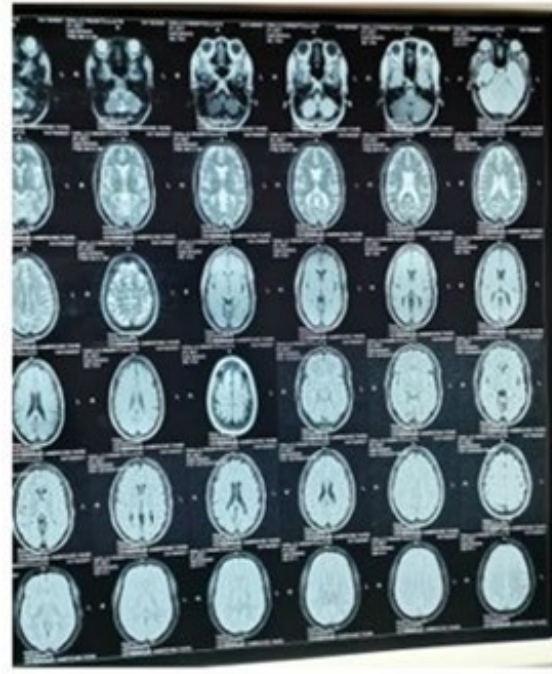


Figure 4: Brain MRI eliminating an expansive intracranial process and other vascular cause.

At the end of the assessments, the diagnosis of HTICI was retained. The patient was placed on a low-calorie diet combined with Acetazolamide 250 mg 2 times daily and corticosteroids (Cortancyl 20mg) with decreasing discontinuation to normalize intracranial pressure.

The evolution was marked by a noticeable improvement in visual signs after 7 days. Headaches and tinnitus, having completely regressed after two weeks of treatment, it was decided to suspend a possible depletive lumbar puncture. Two months after the start of treatment, the fundus and retinography still showed the sequelae of papillary edema; we therefore decided to maintain Acetazolamide and carry out checks every month. The dietary diet is also maintained until a weight loss of at least 10% is achieved.

2.4 Case 2

This was a 12-year-old patient, received in our ward on April 19, 2023. He complained of a sudden drop in visual acuity and tinnitus. The clinical picture was associated with pulsatile headaches localized in the retroorbital region, radiating to the neck and resistant to analgesics. The evolutionary was chronic about 3 years. He had consulted in several structures of the place, without favorable follow-up. The patient had no particular history or known drug allergy. The ophthalmological examination was bilateral and comparative, eye-by-eye and plane by plane.

2.5 To the Right Eye

Visual acuity from afar without correction was 2/10th. With the slit lamp: healthy appendages, transparent cornea; the quiet and deep anterior chamber; the round pupil centered with a photomotor reflex present, IOP measurement was not done (non-cooperation of the patient). Fundus examination after obtaining maximum pupillary dilation revealed stage III papillary oedema (see Figure 5).

2.6 In the Left Eye

Visual acuity from afar without correction was 2/10th. The rest of the ophthalmological examination revealed the same peculiarities of the right eye. In front of these signs, a syndrome of intracranial hypertension was evoked. Retinography and OCT confirmed bilateral stage III papillary oedema (Figure 5 and Figure 6). The CV showed a significant shrinkage above 75% (Figure 7).

The patient was referred to the neurosurgery department where the cerebral MRI performed with T1, T2, T2 FLAIR, diffusion and coronal T2 sequences, returned without particularity to eliminate a circulation disorder-resorption of the CSF, a chronic subdural hematoma and an intracranial expansive tumor process (Figure 8). The lumbar puncture performed, highlighted a clear fluid under pressure and sterile.

He was also sent to the infectious and tropical diseases department where the biological blood tests (complete blood count, retroviral serology and blood biochemistry) carried out were not contributory. This allowed us to eliminate a cerebral and meningeal infection.

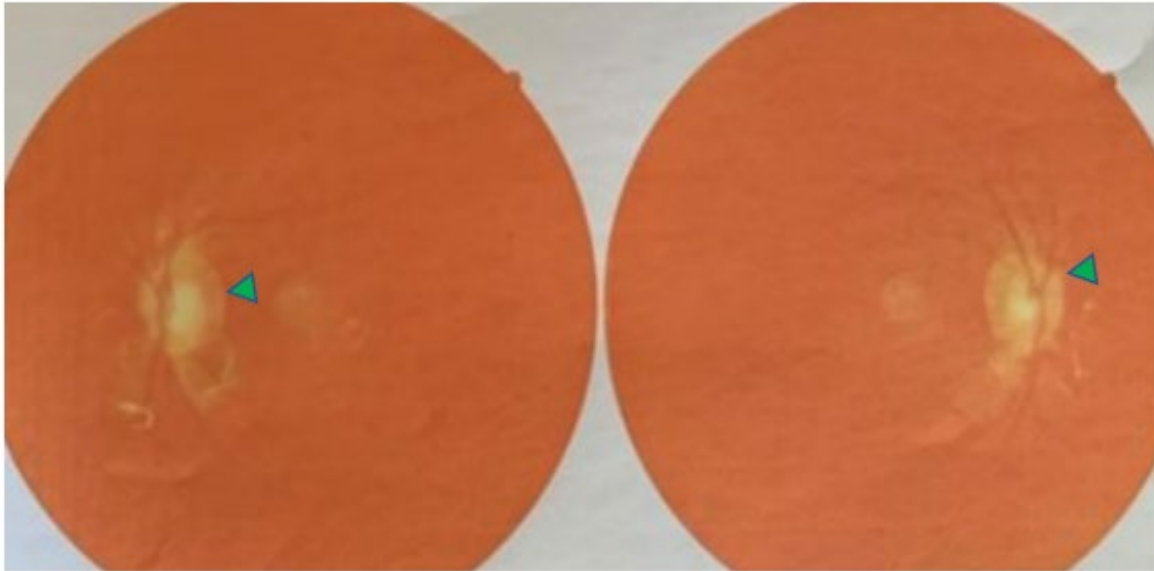


Figure 5: Right and left eye retinography/showing stage III bilateral papillary oedema

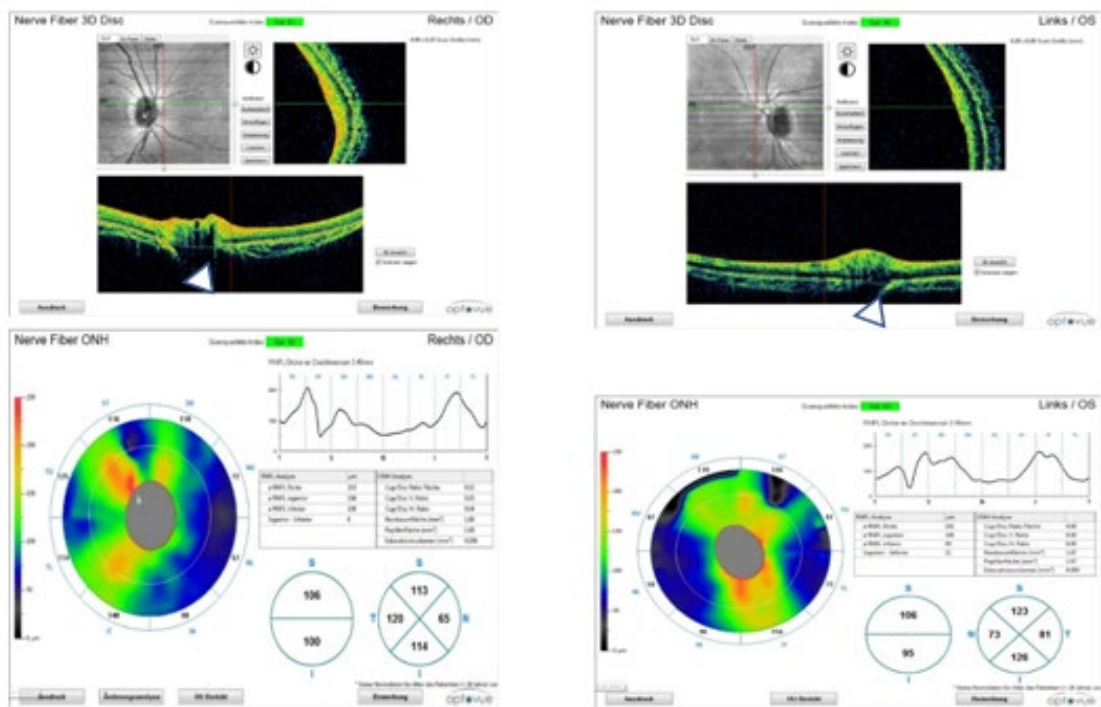


Figure 6: OCT - RNFL Right and left eye, showing bilateral papillary edema stage III.

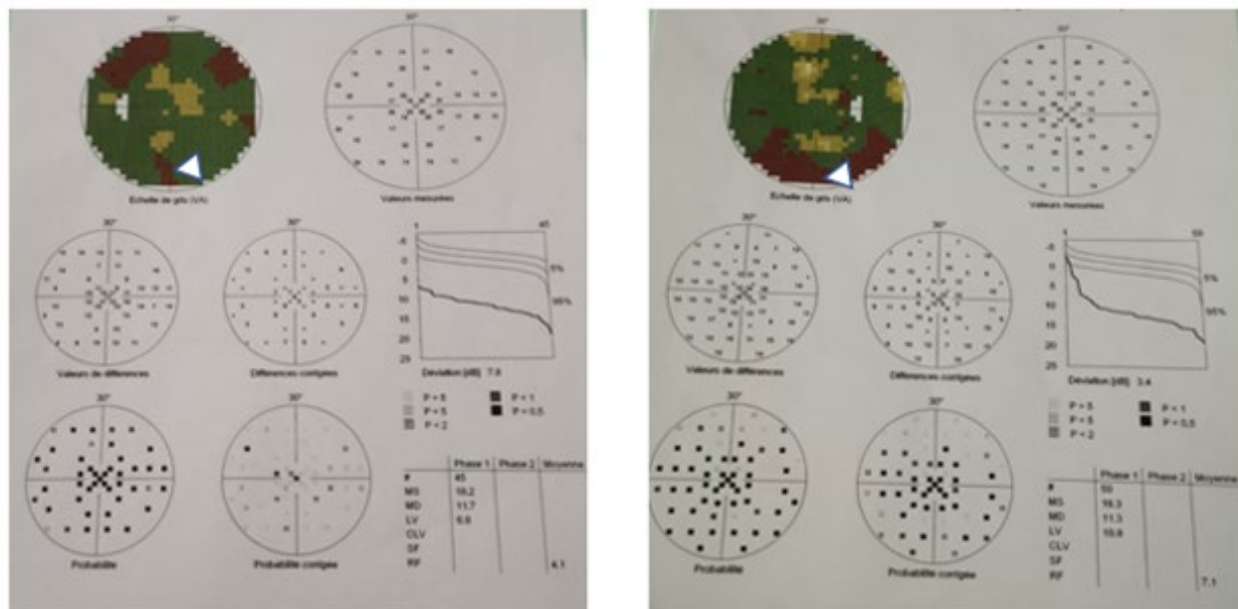


Figure 7: Automated visual field of view right and left eye, showing 75% bilateral narrowing.

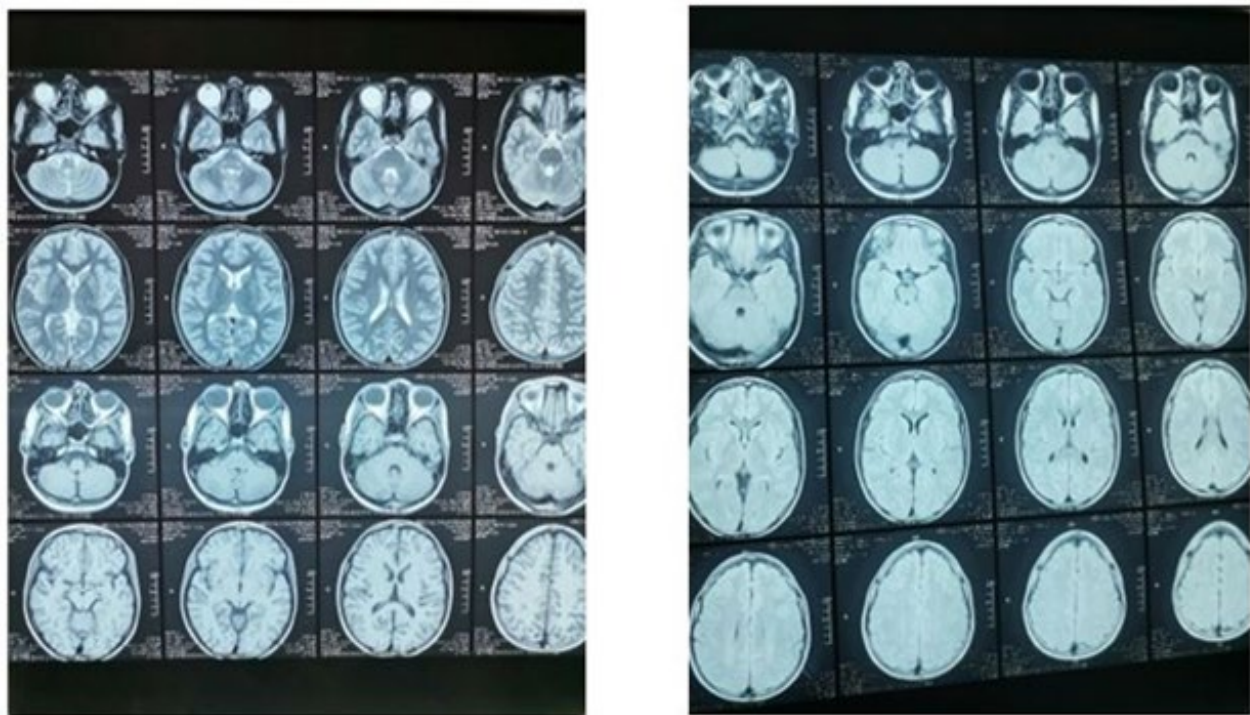


Figure 8: Brain MRI performed showing no particularity

Faced with these bundles of arguments, we retain the diagnosis of idiopathic intracranial hypertension.

The patient was treated with Acetazolamide 250 mg with one (1) tablet 2 times daily.

At two months of treatment, the evolution is marked by a clear improvement in headaches without improvement in visual signs;

however, we decided to refer the patient to Neurosurgery for a possible depletive lumbar puncture and to continue the drug treatment with regular monitoring of visual fields and OCT.

3. Discussions

HICT is an uncommon pathology in the general population with 1

to 2 cases/100,000 inhabitants, but twenty times more common in overweight women [2,3]. This is the case of our overweight patient No. 1. Much more rarely, it affects children in whom the association with obesity is less common like our case No. 2 [4]. Telltale ophthalmological manifestations are headache, visual eclipse and tinnitus [10]. This is the case for our patient No. 2. On the other hand, our patient No. 1 had a visual acuity maintained at 10/10 probably reflecting the integrity of the visual pathways. Papillary edema is the most important sign during Idiopathic Intracranial Hypertension. It is present in all patients and is most often bilateral [11]. Our data corroborate with those of the literature: bilateral papillary edema was found in our two (02) patients respectively in stage I and III confirmed by paraclinical examinations. Brain MRI is the most indicated neuroimaging [12]. The goal of the treatment of idiopathic Intracranial Hypertension is to reduce ICP with the primary objective of preserving vital function and secondarily vision [8]. We introduced a low-calorie diet in patient No. 1, weight loss in two months contributed to the improvement of her clinical condition. The treatment of Idiopathic Intracranial Hypertension is most often medical and relies on Acetazolamide and corticosteroid therapy, although the use of the latter is controversial [13]. Both of our patients received acetazolamide 250 mg in combination with Cortancyl 20mg in patient No. 1. The realization of depletive lumbar punctures is necessary in severe cases of Idiopathic Intracranial Hypertension [14]. We performed a depletive lumbar puncture in patient No. 2. In our study, the evolution was favorable, resulting in a considerable regression of symptoms in the first case. On the other hand, patient 2 was at risk of visual loss due to stage III papillary edema.

4. Conclusion

In front of papillary edema associated with visual disturbances, headaches, tinnitus, it is necessary to suspect HTICI. The positive diagnosis is based on the bundles of epidemiological, clinical and imaging arguments, for which the ophthalmologist plays an important role. Its most formidable ophthalmological complication is irreversible blindness.

Conflicts of Interest

The authors do not declare any conflicts of interest in relation to this work.

Authors' Contributions

Authors contributed to one or more levels of manuscript writing since protocol, data collection, and writing. All have read and approved the manuscript.

Références

1. Smaoui, R., Sakka, S., Daoud, S., Moalla, K.S., & Farhat, N., et al. (2020). Idiopathic intracranial hypertension syndrome and endocrinopathies, a cause-and-effect relationship ? *Rev Neurol (Paris)*, 176, S37.
2. Andrews, L.E., Liu, G.T., & Ko, M.W. (2014). Idiopathic intracranial hypertension and obesity. *Horm Res Paediatr*, 81(4), 217-225.
3. Caroline, Roos. (2016). Intracranial hypertension less and less idiopathic?. *Neurology*, 19(188), 151-155.
4. Andrew, G. Lee., & Michael, Wall. (2021). Idiopathic intracranial hypertension (pseudotumor cerebri): Epidemiology and pathogenesis. <https://somepomed.org/articulos/contents/mobipreview.htm?33/26/34208?view=print> (accessed October 1, 2021).
5. Friedman, D.I., Liu, G.T., & Digre, K.B. (2013). Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. *Neurology*, 81(13), 1159-1165.
6. Elouarradi, H., & Daoudi, R. (2014). Papillary edema of bilateral stasis revealing benign intracranial hypertension. *Pan Afr Med J*, 18, 96.
7. El Ibrahim, F., Baddou, T., Berraho, A., Bengarai, W., & Hajji, Z., et al. (2021). Idiopathic intracranial hypertension: a retrospective study of 16 cases | SFO-online - Société Française d'Ophthalmologie. <https://www.sfo-online.fr/media/18363> (accessed September 29, 2021).
8. Moulaye, A., & Baba, M.J.S. (2020). Idiopathic intracranial hypertension: about two cases. *PAMJ - Clin Med*, 4(47), 1-10.
9. Bidot, S., Biousse, V., & Bousser, M.G. (2014). Idiopathic intracranial hypertension. *EM-Consulte*, 11(1), 1-10.
10. Timmermans, G., Scholtes, F., Andris, C., & Martin, D. (2015). Le Syndrome Pseudotumor Cerebri Primaire ou Hypertension Intracrânienne Idiopathique. *Rev Med Liège*, 70(10), 488-494.
11. Rigi, M., Almarzouqi, J.S., Morgan, L.M., & Lee, A.G. (2015). Papilledema: epidemiology, etiology, and clinical management. *Eye and Brain* 7(1), 47-57.
12. Mvogo, S.E., Enyama, D., Dohvoma, A.V., Njikui, D.N., & Meguize, C.A., et al. (2017). Hypertension Intracrânienne Idiopathique de l'Enfant: À Propos d'un Cas. *Health Sci Dis*, 18(2), 55-57.
13. Riss, I. (2011). Papillary oedema in idiopathic intracranial hypertension. *EMC Neurologie*, 11, 1-10.
14. Thurtell, M.J., & Wall, M. (2013). Idiopathic intracranial hypertension (Pseudotumor cerebri): recognition, treatment, and ongoing management. *Curr Treat Options Neurol*, 15(1), 1-12.

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