

Foster Kennedy Syndrome: About 2 Cases

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Case Report

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Abstract

We relate 2 patients cases whose age are 32 and 35 years affected by Foster Kennedy's syndrom during a 10 years period in Brazzaville (Congo) Teaching Hospital. These two patients had progressive bilateral low visual acuity, papillary edema in one eye and optic atrophgia in another one, in link with pituitary tumor. For all cases, betamethazone-based treatment associated to acetazolamid in bolus by intravenous infusion and per os route has been set up.15 days later we have observed a decline of the papillary edema and the stability of vision acuity. The syndrom of Foster Kennedy is scarcely revealed into the african's literature, the etiological diagnosis refers to computed tomography scan and especially brain magnetic imaging resonance. The etiological treatment remains essential and the prognosis depends onto it.

Keywords: Neuro-Ophtalmology; Foster Kennedy's syndrom

Introduction

The syndrom of Foster Kennedy is a symptom of expansive and compressive process of the anterior cranial fossa which induce a mass effect on tissus and surrounding neurosensory tracts [1]. It is typically characterized by bilateral papillaries anomalies as a papillary atrophgia homolateral to the lesion and stasis papillary in the contrary side of the lesion in inconstant association with anosmia. It has been described by numerous of authors such as: William Gower in 1893, Schultz-Zehden in 1905, Foster Kennedy in 1911 in USA then later the depiction done by Willehm Uththoff in 1915.Regarding the importance of this syndrom caused by cerebral tumor, we relate 2 clinical cases found during a period of 10 years in Brazzaville(Congo) Teaching Hospital.

Goal

Underlining the interest of cerebral imaging accessibility in the diagnosis and neurosurgical treatment of brain tumors.

Observation

First Case

KL, 32 years old, female, visited for the very first time the ophthalmologist. The onset of the illness comes back up about 4 years, by a progressive bilateral low visual acuity, relapsing cephalagias despite taking some antalgic pills of the first stage, and amenorrhoea. During sight exam, we notice on the right eye a low visual acuity quoted 1/10 testifying a papillary edema Stage III, and on the left one, low light reception showing an optic atrophgia (Figure 1). Neurologic exam is normal. We notice in gynecologic

exam an amenorrhea without galactorrhea. The cerebral scan objectified a giant sellar tumor with aggressive appearance, extensive through the left temporal lobe with bone lysis (Figures 2&3).



Figure 1: Left eye optic atrophy (Case 1).



Figure 2: Brain CT-scan in sagittale section showing the sellar tumor (Case1).



Figure 3: Brain CT-scan in coronal section showing sellar tumor with temporal expansion on the right (Case1).

Second Case

ND, 35 years old, male, with no particular ophthalmologic past record, consults for progressive bilateral low visual acuity during 12 months preceded by a bilateral amaurosis. During the sight exam, we notice on the right eye a low visual to the light perception in

keeping with an optic atrophy, on the left eye, a low visual acuity quoted 4/10 in link with a papillary edema stage II (Figure 4). The visual field is off on the right, and it was existing a left nasal hemianopsia (Figure 5). An anosmia has been revealed and neurologic exam was normal. The Scanner showed a hypophyseal tumor which avokes a giant hypophyseal adenoma (Figure 6).



Figure 4: Left eye papillary edema stage II (Case 2).

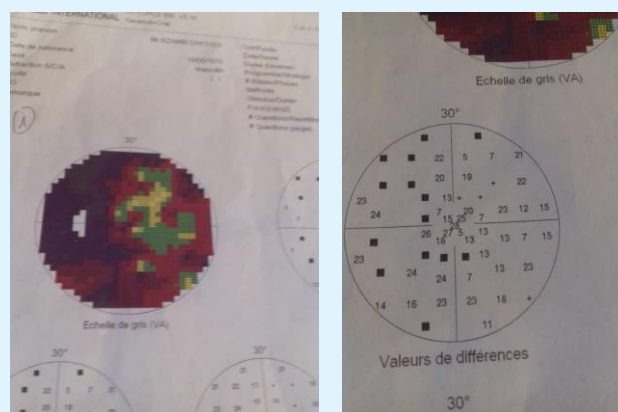


Figure 5: Left side nasal hémianopsia (Case 2).

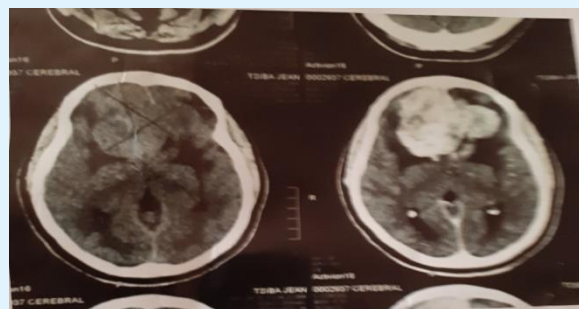


Figure 6: Brain CT-scan in coronal section showing a giant tumor going totally over the pituitary fossa (Case 2).

For these both cases betamethazone-based medical treatment in bolus associated to acetazolamid intravenous infusion and per os route, has been set up while we wait for the intervention of the neurosurgical and anatomopathologist results. 15 days later we have observed decline of the papillary edema and stability of vision acuity. Patients have been lost touch for social and economic reasons.

Discussion

Foster Kennedy's syndrom is scarcely brought in the african's literature. Its prevalence in relation with hypophyseal adenoma is estimated from 0.02 up to 0.1% according to some authors [2]. If the clinical examination is evocative of an endocranial process, the etiological diagnosis refers to computed tomography scan (CT-scan) and especially the brain magnetic imaging resonance (MRI) that provides the best details and constitutes a privilege study [3], a test to determine hormone rate and anatomopathology. The contrast administration within imaging is intimately linked to the level of the endothelio-capillary hyperplasia. The diversity of causes shows that in the 45% of cases we find gliomas, from 10 to 15% we find adenomas, in 15% we have meningiomas and also 15% of neurinomas. The treatment refers precociously to corticoids and carbonic anhydride which proving efficient for intracranial high pressure and papillary edema as noted by Liang and Co [4]. However the

etiological treatment remains essential and the prognosis depends on it. Precisely with appropriate drugs when the tumor is chemosensitive or by a total surgical extraction or partial one, coupled with radiotherapy or radiotherapy itself. The medical imaging is compulsory for treatment control.

Conclusion

The syndrom of Foster Kennedy is a complex disease. The availability of diagnosis measures, treatment and their accessibility are worrying for the Congolese people and most black African countries.

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