

A Silent Corticotropic Pituitary Adenoma with Foster Kennedy Syndrome: A Case Report

El Halouat Nihad^{1*}, Mohcine El Mhadi¹, Bouzidi Adil¹ and Iferkass Said¹

¹Ophthalmology Department, Military Hospital Moulay Ismail Meknès, Morocco.

Authors' contributions

This work was carried out in collaboration among all authors. Author EHN followed the patient and wrote the first draft of the manuscript. Authors MEM and BA wrote the discussion of the study. Author IS managed the literature searches. All authors read and approved the final manuscript.

Article Information

Editor(s):

(1) Dr. Panagiotis Tsikripis, University of Athens, Greece.

Reviewers:

(1) Divyesh N. Kelawala, Anand Agricultural University, India.

(2) Mridula V. Amarnath, Chinmaya Mission Hospital, India.

Complete Peer review History: <http://www.sdiarticle4.com/review-history/62351>

Case Study

Received 14 August 2020
Accepted 19 October 2020
Published 18 November 2020

ABSTRACT

Foster Kennedy Syndrome is unilateral optic disc swelling with contralateral optic atrophy, usually due to a frontal lobe tumour compressing the optic nerve on one side and resulting in papilloedema contralaterally. The etiological and topographical diagnosis uses brain imaging. We describe a case of a silent corticotropic pituitary adenoma associated to a Foster Kennedy syndrome in a 57 years old man. To the best of authors' knowledge, this is the fourth case report regarding pituitary adenoma presenting with Foster Kennedy syndrome. We reviewed the pathogenesis and common clinical manifestations of Foster Kennedy syndrome and would like to highlight the role of neuroimaging techniques in the diagnosis.

Keywords: Foster Kennedy syndrome; optic nerve compression; pituitary adenoma.

1. INTRODUCTION

Foster Kennedy syndrome (FKS) is a rare condition that classically involves optic

nerve atrophy ipsilateral to an intracranial neoplasm with concomitant contralateral papilledema. It usually occurs with fronto-basal tumors, most frequently meningiomas

*Corresponding author: E-mail: elhalouatnihad8@gmail.com, dr.nihadel@gmail.com;

of the sphenoid wing and the olfactory groove [1].

Rarely associated with this syndrome, giant pituitary adenoma can also cause a FKS. In this work, we report a case of a silent corticotrophic giant pituitary adenoma revealed by FKS, review its pathogenesis and highlight the role of neuroimaging in diagnosis.

2. CASE REPORT

A 58 years old male, apparently healthy with no medical history, was presented to our department with a complaint of deep decrease in vision of his right eye accidentally discovered (while occluding contralateral eye), with no other signs. In addition, the patient reports a notion of significant weight gain over 6 months.

On physical examination, the patient had stable vital signs. Ophthalmologic exam revealed visual acuity of no light perception on the right and 7/10 on the left eye. Extraocular movements were normal. There was a right relative afferent pupillary defect. The fundus examination revealed optic atrophy on the right eye (Fig. 1), and marked papilloedema on the left eye (Fig. 2). The Visual field of right eye showed an incomplete temporal hemianopia and enlargement of the blind spot. The neurological examination was normal. He was therefore diagnosed as having the Foster-Kennedy syndrome.

An emergency cerebral resonance magnetic imaging (RMI) showed a polycyclic well limited process of 7 cm in diameter growing up from the sella turcica, compressing the optic chiasma, the 3rd ventricle, invading the sellar floor, the sphenoidal sinus and the right cavernous sinus. Thus, a giant pituitary adenoma was suspected radiologically (Fig. 3). Hence, the patient was referred to neurosurgery department.

Pituitary hormonal profile showed normal levels of serum prolactin level, thyroid hormones, growth hormone and IGF-1. However, ACTH was high: 165 pg/ml [5 – 49 pg/ml], and high morning cortisol 984,6 nmol/l at 8h [171-535 nmol/l].

So, the patient was diagnosed with a giant corticotrophic pituitary adenoma.

The patient underwent a transsphenoidally transnasal endoscopic pituitary surgery.

3. DISCUSSION

In 1911, the neurologist Foster Kennedy described for the first time the Foster Kennedy syndrome (FKS). It is a neuro-ocular syndrome secondary to an intracranial mass effect by an expansive fronto-basal process. FKS is characterized by a papillary atrophy ipsilateral to the lesion and contralateral stasis papilledema.

The FKS is a rare finding. Tonnis [2], in its serie of 3033, histologically verified intracranial tumors, found 28 cases of FKS. Huber [3] saw five cases of FKS among 8100 brain tumors.

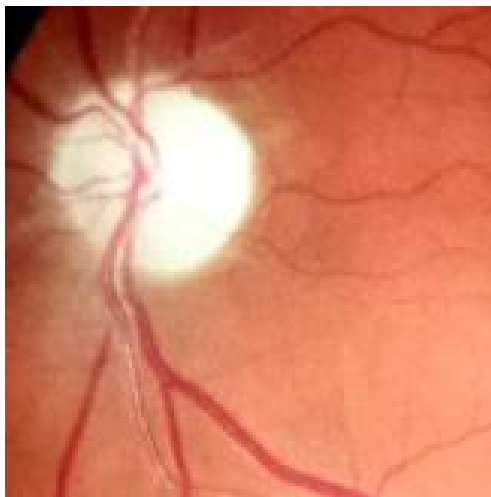


Fig. 1. Fundus of left eye showing pallor of optic disc: Optic atrophy



Fig. 2. Fundus of right eye showing papilledema

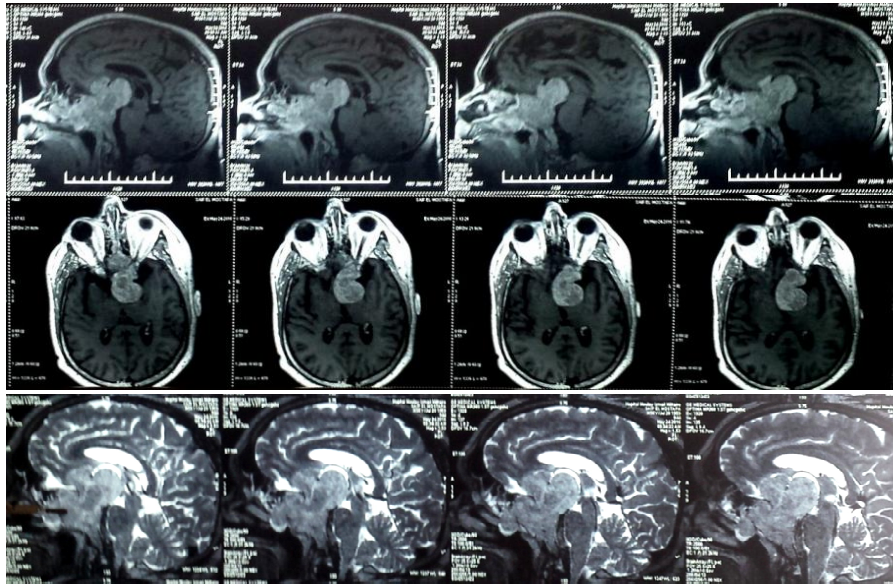


Fig. 3. Cerebral RMI: Giant pituitary adenoma (yellow arrow) well limited of 7 cm in diameter, compressing the optic chiasma, the 3rd ventricle, invading the sellar floor, the sphenoidal sinus and the right cavernous sinus

Through a review of the literature, it was found that this syndrome has been associated with several intracranial frontobasal neoplasm, most frequently sphenoidal ridge meningioma, olfactory groove and suprasellar meningioma [4,5]. Although more commonly associated with neoplasm, the syndrome can also be caused by vascular lesions, meningitis, internal carotid artery sclerosis, and Paget's disease of the skull [5,6].

Pituitary tumors are among the most common intracranial tumors, accounting for 10–15% of all primary intracranial neoplasms in clinical practice [7]. Foster-Kennedy Syndrome has been reported, according to our knowledge, in only three cases as an association.

In 1992, Michael Küchleand [8] published the first case of a non-secreting pituitary adenoma, in a 51 old woman with signs of Foster Kennedy syndrome including right papilledema, left simple optic atrophy, and left anosmia. CT scan and magnetic resonance imaging and endocrinological work-up disclosed a giant non-secreting pituitary adenoma.

In the same year, Simon Ruben [9] reported the second case with a hypersecreting pituitary adenoma in a 27-year-old man. The vision in his both eyes had been declining slowly resulting in complete blindness in this eye and hand

movements on the left. Together with the onset of frontal headaches associated with nausea and vomiting. The fundal examination revealed optic atrophy on the right and marked papilloedema on the left. In addition he had limitation of lateral rectus function bilaterally and limitation of upward gaze. Computed tomography scan of the brain and endocrinological work-up revealed a prolactin-secreting pituitary adenoma.

Biniyam Ayele [10] reported the third case in 2019, in a 21-year old Ethiopian patient, presented with progressive visual disturbance of the left eye, associated with global headache. Neurological examination was pertinent for left side visual loss with optic atrophy and right eye temporal visual field cut with disc edema. Brain MRI and Pituitary hormonal profile showed a prolactin-secreting pituitary adenoma.

The mechanism of optic atrophy and contralateral papilloedema association in FKS cannot be attributed to a single explanation. The hypotheses include [11]: (i) Direct compression of one optic nerve accounts for ipsilateral optic atrophy. Raised intracranial pressure (ICP) causes contralateral papilledema that usually precedes the ipsilateral optic atrophy (ii) bilateral asymmetric optic nerve compression, and (iii) chronically raised intracranial pressure that may lead to early optic atrophy on one side, without direct optic nerve compression.

In our case, the right sided optic atrophy was apparently due to direct adenoma compression to the optic nerve. Contralateral papilledema may have been the result of raised intracranial pressure. The compression of the left optic nerve near the chiasm may also have played a role in the development of papilledema. Additionally, compression of the chiasm might have led to the left temporal visual field defect.

Anosmia and headache are often present in true Foster Kennedy syndrome [1]; however, they are not universally present signs. Other disorders may include emotional lability, memory loss, nausea, vomiting, vertigo, hearing loss, extremity weakness, and facial paresis. Various ophthalmic signs and symptoms may be present depending on the localization of the tumor.

Despite the giant size of the pituitary adenoma the only signs that our patient had were: visual loss in the right eye, temporal hemianopia in the left eye and the rapid weight gain due to hypercosolemia; no other sign were reported.

4. CONCLUSION

Foster Kennedy syndrome is rare but should be recognized. An examination and a search for extra-ophthalmological signs must be carried out. Complementary ophthalmologic examinations make it possible to orient etiologic research, but brain imaging remains essential.

CONSENT

As per international standard or university standard, respondents' written consent was obtained and preserved by the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval was obtained and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Kennedy F. Retrobulbar neuritis as an exact diagnostic sign of certain tumors and abscesses in the frontal lobes. *Am J Med Sci.* 1911;142:355-68.
2. Tonnis W. Diagnostik der intrakraniellen Geschwulste. In: Olivecrona H, Tonnis W: *Handbuch der Neurochirurgie.* Berlin: Springer. 1962;4:1-490.
3. Huber A. Eye signs and symptoms in brain tumors, 3rd Edn. St. Louis: Mosby 1976;171-173.
4. Bynke H. The syndrome of Foster Kennedy. *Acta Ophthalmol.* 1958;36:129-140.
5. Skrzypczak J. Zur bedeutung des Foster Kennedy-syndroms. *Klin Mbl Augenheilk.* 1967;150:504-509.
6. Bhura P, Mishra RK. Foster Kennedy syndrome. *Indian Journal of Ophthalmology.* 1973;21(1):32-33.
7. Mehra KS, Mukerjee KC, Gour KN. Foster-Kennedy syndrome. *Indian Journal of Ophthalmology.* 1965;13(1):39-40.
8. Michael Kuchleand, Hendrikus Seyer. Foster Kennedy syndrome caused by an extensive pituitary adenoma. *Neuro-Ophthalmology.* 2009;12(2):77-83. DOI: 10.3109/01658109209058121
9. Simon Ruben, John Elston, Richard Hayward. Pituitary adenoma presenting as the Foster-Kennedy syndrome. *British Journal of Ophthalmology.* 1992;76:117-119.
10. Biniyam Ayele, Abenet Mengesha, Abdi Wotiye, Yodit Alemayehu. Giant pituitary adenoma presenting with Foster-Kennedy syndrome in a 21-year old Ethiopian a case report. *Ethiop J Health Sci;* 2020. DOI:<http://dx.doi.org/10.4314/ejhs.v30i2.19>
11. Watnich RL, Trobe JD. Bilateral optic nerve compression as a mechanism for the Foster Kennedy syndrome. *Ophthalmology.* 1989;96:1793-8.