Abstract

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A planum meningioma causing a Foster Kennedy syndrome

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Background

Foster Kennedy syndrome (FKS) is a rare neurologic syndrome characterized by ipsilateral anosmia, ipsilateral optic atrophy, and contralateral stasis papilla. It is defined as an orbito-frontal syndrome and can begin with one of the typical symptoms of this syndromic category.

Case history

We describe the case of a 62-year-old female with rheumatoid arthritis on steroid therapy and hypertension that presented to the ED for dysphonic mood, emesis, headache and anosmia. His physical examination was insignificant except for joint deformities and a reduction of bi-temporal hemi field vision. Laboratory tests and EGDS were unremarkable, further evaluation revealed left optic nerve atrophy and right papilledema. Total body CT scan and brain MRI showed an extra cerebral expansive neoformation in the median frontal area adherent to the ethmoid-spheroidal planum compatible with planum meningioma. The patient was therefore referred to neurosurgery for the care continuation.

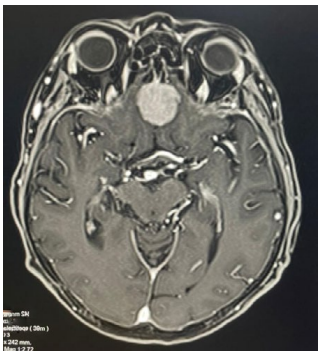

Figure 1. T1-Weighted Contrast Enhanced brain MRI showed an extra cerebral expansive neoformation of 23x11x22 mm in the median frontal brain region, adherent to the ethmoid-sphenoid planum characterized by intense post-contrast graphic impregnation, compatible with planum meningioma.

Discussion

FKS is caused by a intracranial mass by definition, which mechanically compresses the optic nerve and causes atrophy in one eye, and induces elevated intracranial pressure that leads to papilledema in the contralateral eye. The compressing mass/tumor is often an anterior cranial fossa meningioma (this may be in the olfactory groove, sphenoid wing, or frontal lobe), although the middle cranial fossa can also be involved. The ocular disease conditions that have a similar presentation as those seen in patients with FKS include pseudo-foster Kennedy syndrome, non-arteritic anterior ischemic optic neuropathy, and optic nerve hypoplasia, with the classical signs of either unilateral optic atrophy or unilateral papilledema. With regards to diagnosis, FKS requires a detailed case history. The patient should be questioned on vision and sudden reduction or gradual loss of vision. The history should involve probing to elicit the signs of increased intra-cranial pressure, such as headache, diplopia, nausea, and vomiting. Other symptoms usually noticed by patients include anosmia, diplopia, and emotional disorientation. The family members of patients should also be questioned with regards to any changes in the behavior of the patient2. The treatment for FKS tumors includes surgical resection, chemotherapy, radiotherapy and medical therapy (corticosteroids). In our case we supposed that the acute disturbances and psychiatric symptoms were related to the frontal site of the tumor. Physicians should therefore consider FKS in patients presenting for psychiatric conditions, anosmia and changes in vision. A multidisciplinary approach can help to a prompt recognition of the underlying pathology for a better management of FKS.

References

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