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Case report

Case of an isolated oculomotor nerve damage caused by pituitary hemorrhage without cavernous sinus invasion

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ABSTRACT

Introduction: Pituitary apoplexy is a rare endocrine emergency. The term refers to an acute ischemia or hemorrhage into the gland, most often on the basis of preexisting adenoma. The clinical symptoms include a sudden headache, nausea, vomiting, ophthalmic symptoms and hormonal dysfunction. The most severe, life threatening complication of pituitary apoplexy is adrenal insufficiency. The patient may complain of vision disturbances preceded by headaches localized behind the eye. This reflects pressure toward optic nerve caused by expanding mass. When it invades cavernous sinus, it can reach and damage the 3rd, 4th, 5th and 6th cranial nerve. Focal brain ischemia may occur due to direct pressure on internal carotid artery or vasospasm in reaction to local factors.

Aim: To present an example of a clinical evaluation of the patient with an isolated oculomotor nerve damage and comorbidities like diabetes which alone may account for ischemic nerve damage.

Case study: We present a case report of a patient suffering from pituitary apoplexy without cavernous sinus involvement and an isolated oculomotor nerve damage.

Results and discussion: We revise literature on pathophysiology of the third cranial nerve damage in the setting of pituitary apoplexy and make an attempt to explain constellation of the symptoms in our patient.

Conclusions: Pituitary apoplexy should be taken into consideration during a sudden isolated oculomotor nerve palsy. Immediate transsphenoidal pituitary decompression is a potentially effective method of the treatment. In the setting of a little expanding lesion an oculomotor nerve function may be restored without any surgical intervention.

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1. Introduction

Pituitary apoplexy is a rare endocrine emergency.[1] The term refers to an acute ischemia or hemorrhage into the gland, most often on the basis of preexisting adenoma.[2] The clinical symptoms include a sudden headache, nausea, vomiting, ophthalmic symptoms and hormonal dysfunction.[3] Very characteristic are vision disturbances preceded by headaches localized behind the eye.[4] This reflects a pressure toward the optic nerve caused by expanding mass. Moreover, when it invades cavernous sinus, it can reach and damage the 3rd, 4th, 5th and 6th nerves. Internal carotid occlusion may appear due to direct pressure or vasospasm, caused by the factors released from hemorrhagic or necrotic material.[5] This can lead to the focal brain ischemia. Most severe, life threatening complication of pituitary apoplexy is an adrenal insufficiency manifested by e.g. high fever and hyponatremia.[6]

Pathophysiology of pituitary apoplexy is still debated. The anterior pituitary is supplied with blood through a portal system from the infundibulum, which determines incredible perfusion sensibility even to a slight increase of intrasellar pressure.[7] Abnormal angiogenesis in adenoma, which could cause intravascular coagulation and/or make pituitary vessels prone to rupture, was also postulated.[8]

The risk factors of a pituitary apoplexy include male gender, big size of adenoma, hormonally inactive adenoma, anticoagulant and antiplatelet therapy.[9]

We present a case report of a patient suffering from pituitary apoplexy without cavernous sinus involvement and with an isolated oculomotor nerve damage. There are not many reports of such cases in the literature.

2. Aim

To present an example of a clinical evaluation of the patient with an isolated oculomotor nerve damage and comorbidities like diabetes which alone may account for ischemic nerve damage.

3. Case report

A 72-year-old male was admitted to the Neurology Ward due to vision disturbances lasting for the past nine days. The abnormalities included diplopia, escalating when looking left, and left eyelid drooping. The symptoms were preceded by nausea and intense, throbbing headache localized in the orbits. The medical history included diabetes mellitus type 2, hypertension, left peripheral facial nerve damage, and also movement difficulties due to stiffness of limbs and problems with balance. Neurological examination revealed right sided ptosis, exotropia deepening when looking left, right pupil slowly reacting to light, smoothing of left nasolabial fold and weakness of left orbicularis oculi muscle with lagophtalmos (Fig. 1). Moreover, bradykinesia, hypokinesia, resting tremor of the upper limbs along with a cogwheel sign were observed. Pyramidal or meningeal signs were absent. Finger to nose and heel to feet tests along with tandem gait were normal.

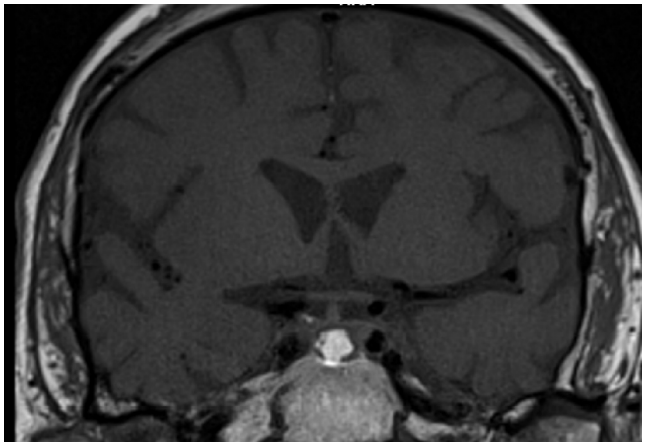

Fig. 2 – Head MRI – pituitary hemorrhage focus. Cavernous sinus invasion not present.

Head MRI revealed hemorrhagic focus in pituitary area with residual fragments of the adenoma, without features of cavernous sinus invasion (Fig. 2). The lesion was hyperintense in both T1 and T2 sequences – estimated age of hemorrhagic focus was 8–14 days. Moreover, in both hemispheres, minor, scattered, old ischemic focuses were present.

Laboratory tests were as follow: Na^+ – 138 mmol/L, K^+ – 4.56 mmol/L, creatinine – 0.9 mg%, HbA1c – 6.8%, TSH – 2.3 UI/L, prolactin – 4 ng/mL, cortisol – 467 nmol/L in at 9 A.M. and 53 nmol/L at midnight. Body temperature was 36.7 °C, heart rate – 70 bpm, blood pressure – 135/80 mmHg.

During hospitalization, the patient was treated with Levodopa. Reduction of intensity of extrapyramidal symptoms

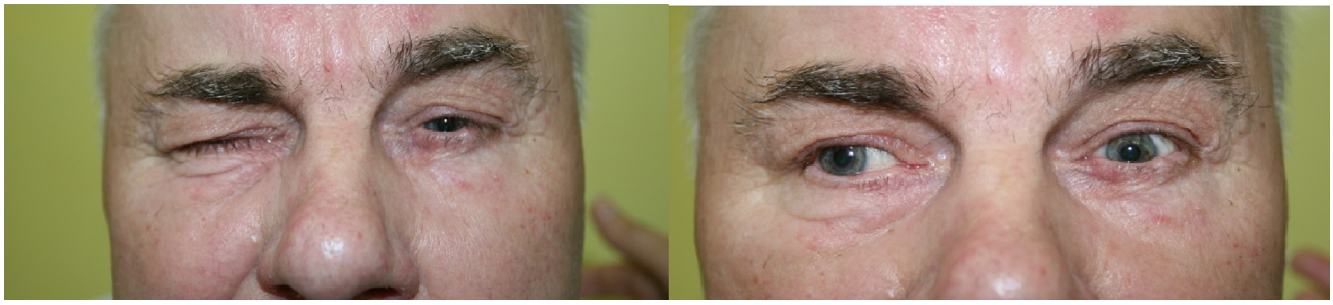

Fig. 1 – In neurological examination: right exotropia (patient looking straightforward), slight right sided mydriasis and left peripheral facial nerve damage.

was observed. Due to fast abatement of ophthalmic symptoms and lack of endocrine complications it was decided not to perform sellar decompression.

At the two month follow-up only discrete weakness of the right eye adduction was observed. Pupillary function was fully restored.

4. Results and discussion

An isolated damage of the oculomotor nerve in pituitary apoplexy is very rare.[10] Pathomechanism in cases when the cavernous sinus is not invaded cannot be clearly defined. Kobayashi et al.[11] described two cases of the patients with an acute hemorrhage to pituitary adenoma. In both cases erosion of posterior clinoid process, caused by adenoma mass was observed in neuroimaging. The authors postulate that during hemorrhage into pituitary gland, an abrupt adenoma expansion toward the input of oculomotor nerve took place.

The entrance site of the 3rd cranial nerve, an oculomotor triangle, constitutes a part of the upper wall of cavernous sinus and it is confined by anterior and posterior petroclinoid and interclinoid ligaments.[12] The oculomotor nerve travels through the upper and lateral portion of caveronus sinus at the level of pituitary gland,[13] initially above posterior petroclinoid ligament, then under interclinoid ligament.[14] In the group of twelve patients with pituitary apoplexy it was stated that the most frequent symptoms of cranial nerves damage referred to the 3rd nerve. The reason for this predilection is the 3rd nerve compression against interclinoid ligament (when progress of the symptoms is slow) or a sudden blood flow disruption through its *vasa nervorum* – a branch of internal carotid artery (in the case of abrupt symptoms onset).[15,16] In a group of the patients with the 3rd nerve palsy and pituitary hemorrhage, with and without ischemic stroke expansion of tumor mass toward interclinoid ligament was demonstrated.[17]

In the case presented in this article the risk factors of vascular disease and sudden onset of the symptoms may suggest limitation of blood supply by *vasa nervorum* as mechanism of the oculomotor nerve damage. Minor pupillary symptoms reflect probably compression component. In differential diagnosis, diabetic neuropathy of the oculomotor nerve was taken into consideration. In the latter usually there is no pupillary involvement. Major abatement of the symptoms was probably caused by hematoma absorption in the pituitary area.

5. Conclusions

Pituitary apoplexy should be taken into consideration during a sudden isolated oculomotor nerve palsy.[10,18] Immediate transsphenoidal pituitary decompression, as a potentially effective method,[11] ought to be considered as a treatment of choice in acute phase of the illness. Nevertheless, in the setting of little expanding lesion the oculomotor nerve function may be restored without any surgical intervention.

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