SPHENOID FISSURE SYNDROME - CASE REPORT

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Abstract: This paper presents a patient with a lower eyelid wound penetrating into the orbit with sphenoid fissure syndrome. There are presented the stages of the diagnosis and the evolution.

INTRODUCTION

Superior orbital fissure (fissura orbitalis superior) is a hole bounded by the small and large wings of the sphenoid bone. Superior orbital fissure is crossed by the oculomotor nerve (the upper and lower branches), trochlear, ophthalmic, abducens, superior ophthalmic vein, sympathetic fibers. Sphenoid fissure syndrome, also known as Rochon - Duvigneaud syndrome, is caused by a damage of the structures crossing the sphenoid slot, manifested by total ophthalmoplegia, which is secondary to the common oculomotor nerve damage, trochlear and external oculomotor, corneal anesthesia by affecting the upper branch of the trigeminal and sometimes exophthalmia by ophthalmic vein compression. There are 3 major causes that lead to the emergence of this syndrome: craniomaxilofacial trauma, orbital tumours: lymphoma, rhabdomyosarcoma, infections or inflammatory diseases.

Superior orbital fissure syndrome of traumatic origin was first described by Herschfeld in 1858. In 1896, Rochon - Duvigneaud described the syndrome as a pathological entity in four patients with syphilis. Classically, sphenoid fissure syndrome is characterized by the paralysis of the three oculomotor nerves which are simultaneously damaged with the ophthalmic nerve. The patient presents: ptosis, ophthalmoplegia, pupil changes, hypoesthesia or anesthesia in the ophthalmic areas, abolished corneal reflex, pain in the area of the ophthalmic nerve, mild exophthalmia (reducible). If the ocular sympathetic fibers are affected, miosis may occur, which is not influenced by sympathomimetic fibers, which indicates the compliance with the ciliary ganglion, and therefore, an extraorbital injury. On the other hand, mydriasis draws attention to an orbital injury with the damage of the ciliary ganglion fibers. It represents fewer than 5 % of cases with painful ophthalmoplegia. It affects equally both genders. It can occur at any age, but mainly in the 5th decade of life.(6,7)

CASE REPORT

We present the case of an 8-year-old patient who is admitted in the Ophthalmology Clinic within the Clinical County Emergency Hospital of Sibiu in 05.03.2013.

The patient, D.L., 8 years old, rural area, presents at the right eye a penetrating palpebral wound, as a result of a punctured trauma with a sharp object (fork). Upon admission, the patient presents on the right eye, decreased visual acuity, ptosis and homonymous diplopia.

Paraclinical and laboratory investigations:

CT skull: Bilateral maxilar and sphenoidal sinusitis.

MRI skull: A slight right exophthalmia, minimal edema in the right retro-orbital fat, without damages of the eyeball, muscles and optic nerve, bilateral maxillary and sphenoidal sinusitis.

Neurological examination: Complete cranial nerve III palsy.

ENT examination: Maxillary sinus infections without bilateral sensibility, nasal cavities without secretion, right ethmoid maxillary rinosinusitis with orbital phlegmon after punctured wound, with superior palpebral ptosis and external oculomotor palsy.

Neurosurgical examination: Cranio-facial trauma. Punctured wound in the right orbit.
After anamnesis, clinical examination and the paraclinical and laboratory investigations, the positive diagnosis is: RE: Sphenoid fissure syndrome. The differential diagnosis of the etiology of sphenoid fissure syndrome was made with: orbital tumours, infections or inflammatory diseases, neurogenic ptosis (common oculomotor nerve palsy - diabetes, aneurysms, brain tumours, strokes etc.), myogenic ptosis (myasthenia gravis, myotonia, myopathy).

Treatment and evolution:
The treatment consisted in large spectrum antibiotics, steroidal and non-steroidal anti-inflammatory drugs, neurometabolic drugs and topical treatment with antibiotics and steroidal and nonsteroidal anti-inflammatory drugs. The evolution under the therapy was favourable with the improvement of neuro-ophtalmological deficits.

The ophthalmological follow up at 1 month showed: VA OD: 20/20 without correction, VA OS: 20/20 without correction, IOP OD = 17 mmHg, IOP OS = 19 mmHg. Posterior pole for both eyes was normal. OD – minimal palpebral ptosis, convergence deviation of the left eye (figure no. 3)

• In our case, the evolution was very good under the therapy with the remission of neuro-ophtalmological signs in 3 months.

REFERENCES