This article describes a case of superior orbital fissure syndrome caused by an internal carotid artery aneurysm in the cavernous sinus. Etiology, clinical presentations, and diagnostic methods are discussed. Possible regression of signs and symptoms after timely endovascular treatment of an internal carotid artery aneurysm in the cavernous sinus is reported.

**Key words:** superior orbital fissure syndrome; internal carotid artery aneurysm; cavernous sinus; endovascular stent assisted coiling.

**INTRODUCTION**

The superior orbital fissure (SOF) syndrome is represented by a symptom complex involving disorders of cranial nerve (CN) pairs III, IV, V, and VI, which pass through the SOF. Hirschfeld first described the SOF syndrome in 1858 in a patient with retrobulbar pain, paresis of the oculomotor nerve, and disorder of tactile sensitivity in the innervation zone of the branches of n. ophthalmicus due to post-traumatic hematoma located near the SOF. In 1896, Rochon-Duvigneaud reported the SOF syndrome in four patients with syphilitic periostitis presenting with similar symptoms [12]. In 1962, Lakke described the currently accepted definition of the SOF syndrome, which included ophthalmoplegia, ptosis, mydriasis, exophthalmos, and a disorder of tactile sensitivity in the innervation zone of the first branch of the trigeminal nerve [11].

The main causes of the SOF syndrome include craniocerebral trauma, cavernous sinus hematoma,
syphilitic periostitis, neoplasms, aneurysm of the cavernous segment of the internal carotid artery (ICA), or a cavernous arteriovenous fistula. In cases where the listed reasons are excluded, it is customary to consider an idiopathic process. Regardless of the etiology, the clinical manifestations of the SOF syndrome are similar and result from inflammation or compression of the corresponding anatomical structures [16]. In a number of cases, the SOF syndrome can be partial, which is determined by the degree of compression and its involvement in the pathological process of individual nerve trunks passing through the fissure.

An ICA aneurysm in the cavernous sinus is observed in 2%–9% of all intracranial aneurysms and 15% of ICA aneurysms [2, 3, 20]. The SOF syndrome most often develops from an infrasphenoid (below the sphenoid processes of the Turkish saddle) or suprasphenoid location of the ICA aneurysm. We report a case in which the SOF syndrome was caused by an ICA aneurysm in the cavernous sinus.

**DESCRIPTION**

Patient K., aged 54 years, presented to an ophthalmo-oncologist in city general hospital No. 2 in December 2015 with complaints regarding drooping of the upper right eyelid, diplopia, and periodic pressing pains in the right frontal and paraorbital regions that arose a month earlier. On examination, visual acuity was 0.6 sphere + 0.75 = 0.8 in the right eye (OD) and 0.8 sphere + 0.75 = 1.0 in the left eye (OS). Intraocular pressure measured according to the Maklakov method was 20 mm Hg in both the eyes. The visual field, determined with kinetic perimetry was normal.

Clinical examination of OD revealed that the upper eyelid drooped and completely covered the pupil. The eyeball was deviated outward. The Hirschberg squint angle was 25°. According to exophthalmometry, the right eye was proptotic up to 3 mm compared with the left eye. Movement was limited upward, downward, and inward while maintaining abduction. The pupil was round, centered, and dilated to 5 mm in diameter; it showed no direct reaction to light. In the OD ocular fundus, the optic nerve disc (OND) was pale pink with distinct contours. The cup/disc ratio was 0.3, and the vascular bundle was in the center. The stroke and caliber of the vessels were unchanged. No pathology was observed in the macular zone and on the periphery. Clinical findings of OS were within normal limits.

Based on the objective data, the SOF syndrome was diagnosed. Magnetic resonance imaging (MRI) of the brain and orbital cavities was performed to clarify the diagnosis. MRI of the orbital cavities revealed a 13 × 11 mm saccular aneurysm at the site of the right posterior communicating artery (RPCA) origin from ICA without signs of thrombosis (Figs. 1, 2).

Contrast-enhanced multispiral computed tomography (CT) of the brain confirmed the presence of a large 15 × 11 × 11 mm aneurysmal enlargement of ICA in the lower posterior wall in the immediate vicinity of RPCA entrance. The neck of the aneurysm

---

**Fig. 1.** MRI brain angiogram demonstrating a 13 × 11 mm saccular aneurysm (arrow) without intramural thrombus at the place of divergence of posterior communicating artery from internal carotid artery

**Рис. 1.** МРТ головного мозга. Мешотчатая аневризма (указана стрелкой) в месте отхождения правой задней соединительной артерии от внутренней сонной артерии размером 13 × 11 мм без признаков тромбоза

**Fig. 2.** MRI brain angiogram. Horizontal section of saccular aneurysm of internal carotid artery (arrow)

**Рис. 2.** МРТ головного мозга. Горизонтальный срез. Локализируется мешотчатая аневризма внутренней сонной артерии (указана стрелкой)
was 3 × 4 mm in size. Because of the nature of the aneurysm (common unruptured), intravascular embolization of the communicative segment of RPCA was performed with detachable microcoiling and stent assistance (Almazov Federal Medical Research Center). There were no complications during the surgery.

At the follow-up ophthalmological examination conducted a month after intravascular embolization of the cavernous ICA aneurysm, the visual acuity of OD was 0.7 sphere + 0.75 = 0.8. Intraocular pressure measured according to the Maklakov method was 20 mm Hg. The kinetic perimeter parameters were normal. The right upper eyelid was lowered, with the lower edge covering the middle of the pupil. The eyeball was turned laterally. The Hirschberg squint angle was 30°. Movement of the eyeball was limited upward, inward, and moderately downward; complete abduction was maintained. The pupil was round, centered, and dilated to 5 mm in diameter; it showed no direct reaction to light. In the OD ocular fundus, OND was pale pink with distinct contours, the cup/disc ratio was 0.3, and the vascular bundle was in the center. The stroke and caliber of the vessels were unchanged. No pathology was observed in the macular zone and on the periphery.

The patient is under observation in the City Hospital No 2.

**DISCUSSION**

The SOF is formed by the body of the sphenoid bone and its wings, and it connects the orbit with the middle cranial fossa [19]. It is pear-shaped and has a long axis, which is medially directed at an angle of 45° from a wide base to a laterally located apex. In 2008, based on the autopsy data of 100 skulls, Reymond et al. detected two morphological types of SOF: type A shows typical narrowing; whereas type B does not. SOF is significantly shorter in type B than in type A [18].

The dimensions of SOFs are 2–3 × 22 mm². The tendon of the lateral rectus muscle divides the SOF into two parts: the upper part, which includes CN IV, the frontal and lacrimal nerves, and the upper branch of the ophthalmic vein, and the lower part, which is enclosed in the tendon ring and consists of the upper and lower branches of the oculomotor nerve, CN VI, the nasociliary nerve, and the lower branches of the ophthalmic vein.

One reason for development of the SOF syndrome, as mentioned above, is the ICA aneurysm in the cavernous sinus. This can be an accidental finding on neuroimaging and is defined as asymptomatic [1]. When increasing in size, the saccular enlargement of ICA can maintain its integrity (the so-called unruptured aneurysm, showing continued blood flow with symptoms of compression of adjacent anatomical structures) or can rupture into one of the three spaces. Pronounced, sometimes fatal, epistaxis indicates a rupture in the medially located wedge-shaped sinus. Due to the extradural position, the risk of subarachnoid hemorrhage (SAH) is low and is determined by the size of the aneurysm; for aneurysms with a diameter of <12 mm, the 5-year probability of SAH is 0% versus 3.0% at a diameter of 13–24 mm and 6.4% at a diameter of >25 mm. The rupture of an aneurysm in the cavernous sinus leads to the formation of a direct carotid-cavernous fistula [9, 13, 14, 22].

In 5% of cases, a carotid aneurysm in the cavernous sinus causes the SOF syndrome by compressing the vessels passing through the cavernous sinus and CNs. Ptosis is caused by the disorder of sympathetic fibers of the cavernous sinus, which leads to a loss of Muller’s muscle tone, as well as the involvement of somatic efferent fibers of the oculomotor nerve, which causes dysfunction of the surface and deep plate of the upper eyelid levator. Ophthalmoplegia is caused by compression of the centrally located cavernous sinus of CN pair VI and CN pairs III, IV, and V, adjacent to the lateral wall of the sinus. The absence of a corneal reflex is caused by an abnormality in the transfer of impulses along the nasociliary nerve. Involvement of the parasympathetic fibers of the oculomotor nerve manifests as paralysis of the ciliary muscle, which is manifested in the form of dilatation of the pupil and absence of a direct reaction to light and accommodation. Propptosis is caused by a loss of the tone of the extraocular muscles, which normally have a retractile effect on the eye. A lesion on the lacrimal and frontoab branches of the trigeminal nerve leads to a loss of sensitivity in the forehead and upper eyelid area. Decreased visual acuity indicates involvement of the optic nerve in the pathological process and development of the orbit apex syndrome [7, 14, 17].

ICA aneurysms in the carotid sinus are diagnosed using structural neuroimaging methods. The gold standard tests are CT angiography and digital subtraction cerebral angiography (DCA), which enable detection of very small aneurysms (from 1.3 mm in diameter), to perform 3D reconstruction and processing of the obtained data [15].

Indications for surgical treatment include ruptured as well as unruptured large (16–25 mm) and giant (>25 mm) ICA aneurysms in the carotid sinus [5, 6, 10, 21]. Currently, two main methods are used to exclude aneurysms from the blood flow: open microsurgical operations and endovascular interventions.
Initially, after craniotomy, incision of the dura mater and arachnoidal dissection are performed, with isolation of the main vessels at the base of the brain and aneurysm. The aneurysm is excluded from the blood flow by clipping the aneurysm neck. Endovascular methods to treat ICA aneurysms in the carotid sinus have become widespread. They include the use of latex cylinders, occlusion of aneurysms with the help of detachable microcoils or embolizing materials, and endovascular stent-assisted coiling.

Based on the results of numerous observations, surgical treatment can result in complete elimination or less prominence of SOF symptoms. In this case, the symptoms may persist because with endovascular interventions, embolization of the aneurysm cavity occurs while maintaining its dimensions and consequently leads to its compression. Because of the absence of pulsation of the saccular extension, the symptoms can be reduced. However, cases of progression of SOF symptoms with the presence of previous compression neuropathy have been reported [4, 8].

CONCLUSION

Establishment of the presence of symptoms characteristic of the SOF syndrome in patients requires MR angiography, CT angiography, and DCA to determine the reasons for the last application of structural neuroimaging methods with the possibility of evaluating the arterial blood flow. Timely implementation of adequate surgical intervention leads to a reduced risk of dangerous complications caused by the rupture of aneurysms. The main criteria for determining the treatment approach are the data on the nature and size of the aneurysm.

REFERENCES


Information about the authors:

Yuriy S. Astakhov — MD, doctor of medical science, professor. Department of Ophthalmology. FSBEI HE I.P. Pavlov SPbSMU MOH Russia, Saint Petersburg, Russian Federation. E-mail: astakhov73@mail.ru.

Ol’ga A. Marchenko — ophthalmologist. City Ophthalmologic Center of City hospital No 2, Saint Petersburg, Russian Federation. E-mail: oamarchenko@yandex.ru.

Vitaly V. Potemkin — PhD, assistant professor. Department of Ophthalmology of the FSBEI HE I.P. Pavlov SPbSMU MOH Russia, Saint Petersburg, Russian Federation. E-mail: potem@inbox.ru.

Aleksandra I. Titarenko — resident. Department of Ophthalmology of the FSBEI HE I.P. Pavlov SPbSMU MOH Russia, Saint Petersburg, Russian Federation. E-mail: titarenko@yandex.ru.

Сведения об авторах:

Юрий Сергеевич Астахов — д-р мед. наук, профессор кафедры офтальмологии. ФГБОУ ВО ПСПбГМУ им. И.П. Павлова Минздрава России, Санкт-Петербург. E-mail: astakhov73@mail.ru.

Ольга Анатольевна Марченко — врач-офтальмолог. СПб ГБУЗ «Городская многопрофильная больница № 2». Санкт-Петербург. E-mail: oamarchenko@yandex.ru.

Виталий Витальевич Потёмкин — канд. мед. наук, доцент кафедры офтальмологии. ФГБОУ ВО ПСПбГМУ им. И.П. Павлова Минздрава России, Санкт-Петербург. E-mail: potem@inbox.ru.

Александра Ивановна Титаренко — клинический ординатор, кафедра офтальмологии с клиникой. ФГБОУ ВО ПСПбГМУ им. И.П. Павлова Минздрава России, Санкт-Петербург. E-mail: titarenko@yandex.ru.