CAROTID-CAVERNOUS FISTULA FROM THE PERSPECTIVE OF AN OPHTHALMOLOGIST – A REVIEW

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Sworn declaration
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SUMMARY

Carotid-cavernous fistula (CCF) is an abnormal communication - vascular connection between arteries and veins in the cavernous sinus. Classification according to etiology is traumatic vs spontaneous. According to blood flow rate per high flow vs low flow fistula. According to anatomy of direct vs indirect: Direct (direct) CCF arises through direct communication between the internal carotid artery (ICA) and the cavernous sinus. Indirect CCF originates through indirect communication through the meningeal branches of ICA, external carotid artery and cavernous sinus (not directly with ICA) and Barrow type A, B, C, D division. Patient's subjective complaints depend on the type of CCF. Most often it is pulsating tinnitus, synchronous with blood pulse. Typical findings include protrusion and pulsation of the eyeball, corkscrew vessels - arterialization of conjunctival and episleral vessels, increased intraocular pressure, not responding to local antiglaucomatous therapy, keratopathy a lagophthalmo, corneal ulcers. In the later untreated stages of CCF, secondary, venous stasis or central retinal vein occlusion can occur. Diagnostic procedures include B-scan and color Doppler ultrasonography, digital ophthamodynamometry, computer tomography, nuclear magnetic resonance and digital subtraction angiography. CCF can simulate orbitopathy, conjunctivitis symptoms, carotid occlusion, scleritis or cavernous sinus thrombosis. The ophthalmologist should recognize and indicate the necessary examinations in a timely manner. The therapy is ophthalmological, neuroradiological, sterotactic, surgical and conservative.

Key words: carotid-cavernous fistula; cavernous sinus; caput medusae; corkscrew vessels; proptosis; venous stasis; exophthalmos; ocular pathology; ultrasonography

INTRODUCTION

Carotid-cavernous fistula (CCF) is an abnormal communication – vascular connection between the arteries and veins in the region of the cavernous sinus (sinus cavernosus – SC). From a topographical perspective, the SC is a venous network of a spongiform character. The ophthalmic vein (vena ophthalmica) flows into the SC, and the internal carotid artery (arteria carotis interna – ACI) and the abducens nerve (nervus abducens) pass through the SC. The ophthalmic and maxillary branches of the trigeminal nerves (nervi trigemini), trochlear nerve (nervus trochlearis) and oculomotor nerve (nervus oculomotorius) are located on the periphery of the SC (Fig. 1).

EPIDEMIOLOGY

From an epidemiological perspective, CCF is a rare pathology. It occurs in 0.2 % of patients with craniocerebral trauma [1] and as many as 3.8 % patients with basilar skull fracture [2]. Non-traumatic CCF occurs more frequently in women and in middle to advanced age [3]. In the overwhelming majority fistulas are unilateral, but bilateral CCFs have also been described in the literature [4,5].

ETIOPATHOGENESIS

The most common cause (70–90 %) of CCF is trauma [6] in the intracranial or periorbital region. Direct carotid-cavernous fistulas are distinguished by a direct connection between the intracavernous segment of the

internal carotid artery and the cavernous sinus. These fistulas usually have a high through-flow of arterial blood, and are most frequently caused by a traumatic defect in the arterial wall.

Approximately 24 % of CCFs occur non-traumatically – spontaneously. In the case of indirect CCF, a communication is established between the SC and one or more of the meningeal branches of the ACI, the external carotid artery (arteria carotis externa – ACE) or both the ACI and ACE. These fistulas usually have a low – slow through-flow. We encounter these fistulas most frequently in the case of disorders of the conjunctival tissues (Ehlers-Danlos syndrome, collagenosis), upon damage to the vascular wall of the carotid artery (aneurysm, dissection), upon hypertension and cerebral arteriosclerosis [7], upon atherosclerosis or in the case of rupture of an aneurysm. Opinions differ on the pathogenesis of indirect CCFs. Defects in the vascular walls occur upon a background of congenital arteriovenous malformations, which develop spontaneously or in connection with atherosclerosis, systemic hypertension, collagen vascular disease, pregnancy, and during or after childbirth. In an extensive study by Debrun [9], in which 132 patients with classification according to Barrow were examined, 75.8 % were classified as type A fistulas, 3 % as type C fistulas and 21.6 % as type D fistulas.

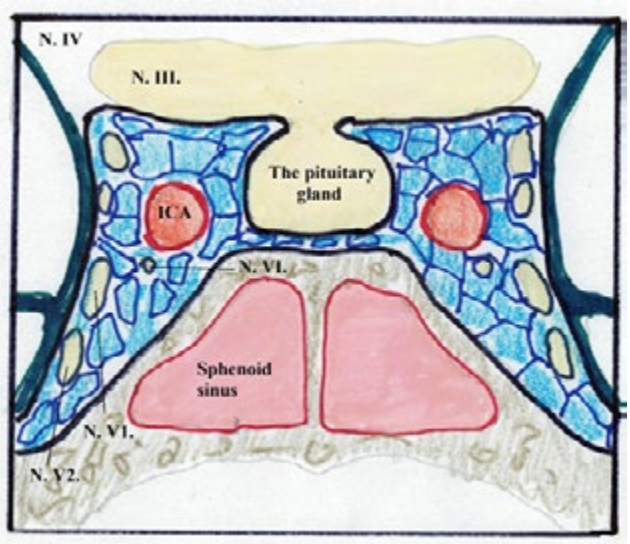

Fig. 1. View of cavernous sinus
ACI – internal carotid artery. N. III – oculomotur nerve. N. IV – trochlear nerve. N. VI – abducens nerve. N. V1 – ophthalmic branch of trigeminal nerves. N. V2 – maxillary branch of trigeminal nerves.

A minimal percentage of CCFs originate iatrogenically – during surgical procedures. For example, following a surgical procedure in the region of the pituitary gland, after therapy for neuralgia of the trigeminal nerve, thrombectomy of the carotid artery, during operations on the ethmoid sinuses etc. [8].

Classification:

- According to etiology as traumatic vs. spontaneous.
- According to speed of blood flow as high-flow vs. low-flow fistulas.
- According to anatomy as direct vs. indirect: Direct CCF originates through direct communication between the internal carotid artery and the cavernous sinus. Indirect CCF originates through indirect communication through the meningeal branch of the ACI, ACE and cavernous sinus (thus not directly with the ACI).
- More precise classification is according to Barrow [10] into four types: Barrow type A – direct CCF: communication between the cavernous sinus and ACI. Barrow type B – indirect CCF: dural connections between the cavernous sinus and meningeal connections of the ACI. Barrow type C – indirect CCF: dural connections between the cavernous sinus and meningeal branches of the ACE. Barrrow type D – indirect CCF: communication between the cavernous sinus and meningeal branches of the ACI and ACE. The classification is presented synoptically in table 1.

Subjective complaints

Subjective complaints, similar to objective findings, are highly influenced by whether this concerns direct CCF (Barrow type A) or indirect CCF (Barrow types B,C,D). Subjective complaints of direct CCF are above all pulsating (subjectively disruptive) tinnitus, synchronous with blood pulse. Headache is generally non-spe-

Table 1. Classification of carotid-cavernous fistula

Classification of CCF according to:	Etiology	Speed of blood flow	Anatomy	Barrow
	Traumatic	High flow	Direct	**Barrow type A:** direct CCF: communication between SC and ACI
	Non-traumatic, or spontaneous	Low flow	Indirect	**Barrow type B:** indirect CCF: dural connections between SC and meningeal connections of ACI
				Barrow type C: indirect CCF: dural connections between SC and meningeal branches of ACE
				Barrow type D: indirect CCF: communication between SC and meningeal branches of ACI and ACE

cific, mostly frontal and ipsilateral with the fistula, sometimes linked with paresthesia to pains in the face. In the case of direct CCF, patients state retrobulbar pulsating pressure. In the case of indirect CCF (Barrow types B, C, D) pulsating tinnitus is minimal, occasional or absent, and is not subjectively disruptive. Patients not infrequently complain of deterioration of central visual acuity and diplopia. However, patients rarely notify a doctor of these complaints, and as a result it is necessary to ask actively about any pulsation and tinnitus within the framework of differential diagnostics.

DIAGNOSIS

Ophthalmological diagnosis:

When damage occurs to the intracavernous segment of the ACI, ACE and their branches, the arterial blood from these arteries flows directly into the venous SC. Increased venous pressure in the SC causes dilation of the superior ophthalmic vein (vena orbitalis superior – VOS) and a deterioration of venous outflow from the eye and eye socket. At the same time, perfusion pressure in the ophthalmic artery decreases, which may lead to retinal ischemia and malfunction of vision. In addition to this, increased venous pressure in the SC may lead to a compression of the surrounding tissues such as the n.VI, n.IV, n.III and n.V1 – ophthalmic branch of the trigeminal nerves and the n.V2 – maxillary branch of the trigeminal nerves. This compression may be manifested for example as ophthalmoplegia [11]. On a background of the above-stated pathological changes, ophthalmologists may encounter a broad range of pathological changes in the eye and eye socket. These may sometimes resemble other ocular diseases – stated in differential diagnostics below. Direct CCF always has more fruste ocular manifestations than indirect CCF.

Typical symptoms encountered by ophthalmologists are:

- Pulsating murmur ipsilaterally with CCF in the periorbital region. Protrusion of eyeball, almost always unilateral (Fig. 2).
- Pulsation of eyeball (visible or detectable palpably). This always appears in direct CCF, sometimes in indirect CCF.
- Caput medusae: corkscrew vessels – arterialisation of conjunctival and episcleral blood vessels (Fig. 3, 4).
- Chemosis of conjunctiva + tumescence of eyelids (Fig. 5).
- Increased intraocular pressure (IOP), not responding to local anti-glaucomatous therapy.
- We encounter edema of the optic nerve disc only in the case of direct CCF [12].
- Diplopia: n.VI is most often afflicted due to anatomical flow in centre of SC.
- Depending on damage to n.III ptosis of upper eyelid, pupillomotor dysfunction.
- Keratopathy and lagophthalmos, corneal ulcers.
- Haemorrhage into vitreous body, retina.

In late untreated stages of CCF neovascular glaucoma, secondary angle-closure glaucoma (after a longer time increased orbital pressure leads to congestion of the iris) may occur [13,14]. Venous stasis causes deterioration of the retinal blood flow, which may lead to central retinal vein occlusion [15]. In one clinical trial, a "3 point signal" was demonstrated to aid timely diagnosis of CCF: hyperaemia of the disc of the optic nerve (DON) + dilation of retinal vein and haemorrhage into retina [16].

For more precise specification of the diagnosis it is suitable also to indicate the following examinations:

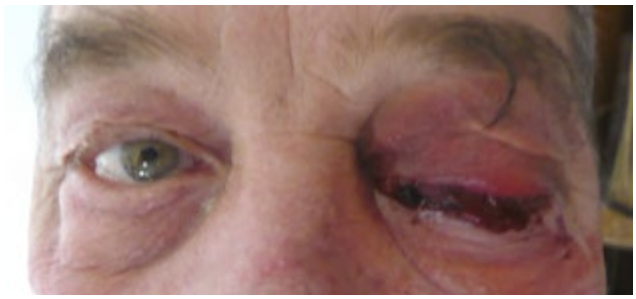

Fig. 2. Ipsilateral protrusion of eyeball upon direct carotid-cavernous fistula in left eye

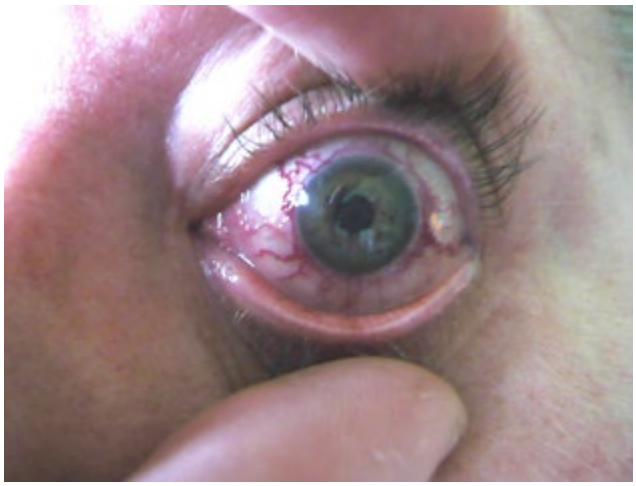

Fig. 3. Caput medusae: corkscrew vessels – arterialisation of conjunctival and episcleral blood vessels in left eye upon direct carotid-cavernous fistula

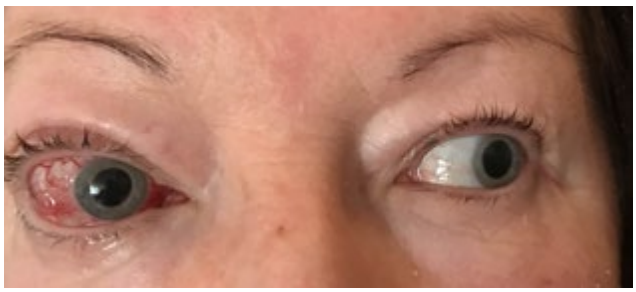

Fig. 4. Caput medusae: corkscrew vessels – arterialisation of conjunctival and episcleral blood vessels in right eye upon direct carotid-cavernous fistula

- **Digital ophthalmodynamometry:** CCF increases pressure in the central retinal vein, as well as episcleral pressure. Following endovascular therapy these pressure parameters are normalised. Digital ophthalmodynamometry reliably and directly enables measurement of pressure in the central retinal vein, and approximate intracranial pressure [17].
- **Ultrasonography (USG):** B-scan displays dilated VOS (Fig. 6), slightly enlarged extraocular muscles (EOM) by approx. 1–3 mm ipsilaterally, contralaterally the EOMs are generally within the norm. Colour Doppler Ultrasonography (CDU: display of colour retrograde blood flow in the VOS in comparison with the other eye (Fig. 7). Typical findings are pathological decrease of flow speed in the central retinal artery ipsilaterally in comparison with the other eye (Fig. 8) as well as enlarging of the resistance index in the central retinal artery ipsilaterally in comparison with the other eye.

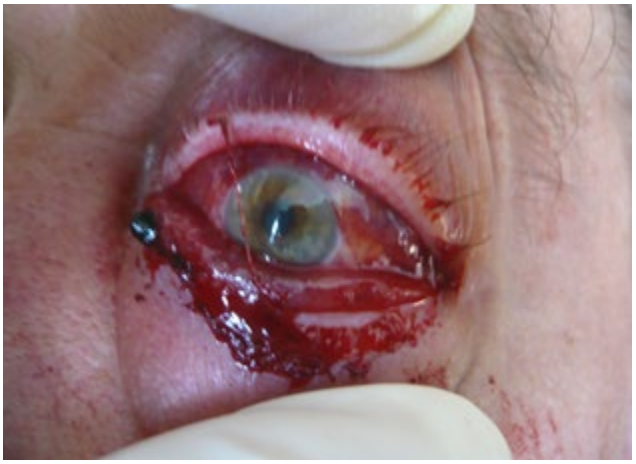

Fig. 5. Pronounced extraocular haemorrhage, fruste chemosis with subconjunctival bleeding and edema of the eyelids upon direct carotid-cavernous fistula

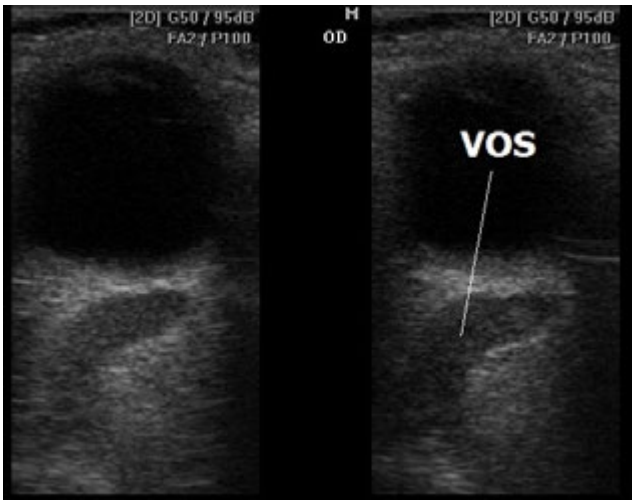

Fig. 6. Dilation of echo of superior ophthalmic vein (vena orbitalis superior – VOS) with the aid of ultrasonic B-scan upon carotid-cavernous fistula

- **Radiological diagnosis:** With regard to the fact that nuclear magnetic resonance (NMR) provides a better display of vascular structures than computer tomography (CT), it is suitable to indicate NMR of the brain, focusing on the region of the SC. NMR without angiography may demonstrate only dilation of the extraocular muscles, dilation of the VOS and potentially also enlargement of the damaged area of the SC [18]. For this reason, in the case of suspicion of CCF it is suitable to indicate NMR with contrast (NMR-AG). This is very important information for the radiologist with regard to suspicion of possible CCF.

Despite the fact that CT, NMR and NMR-AG are useful, they do not determine CCF [19]. In some cases NMR-AG does not display the fistula, despite the presence of CCF [20]. Specialised ophthalmological examinations such as digital ophthalmodynamometry, USG of the eye and orbit, including Colour Doppler examination and CDI, NMR, NMR-AG, serve as a "springboard" for the radiologist, neurologist and neurosurgeon. Within the framework of mutual co-operation, similarly as in the case of intracranial hypertension [21], intracerebral digital subtraction angiography (DSA) is subsequently indicated [22]. Although DSA is an invasive test which today has minimal complications, such as thrombosis, cerebral vasospasm, nerve damage and haemorrhage, it still represents the gold standard for the diagnosis of CCF.

Differential diagnosis

CCF has a broad range of clinical manifestations. As a result it is important to consider also other clinical findings, or conversely, in the case of the changes stated below, also to consider the possibility of CCF within the framework of differential diagnostics.

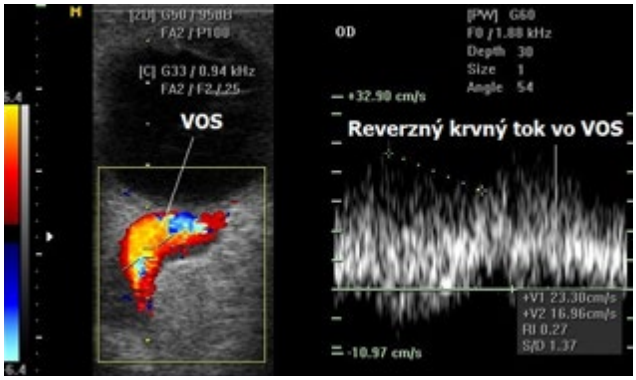

Fig. 7. Colour display of retrograde flow and dilation of VOS upon direct carotid-cavernous fistula with the aid of Colour Doppler ultrasonography. Left image: Under physiological conditions the venous blood flow is coded blue, and the arterial flow red. In the case of carotid-cavernous fistula, retrograde blood flow ensues, meaning a change of the colour coding from blue to red. Right image: recording of spectral analysis of retrograde blood flow of VOS – reverse blood flow in VOS

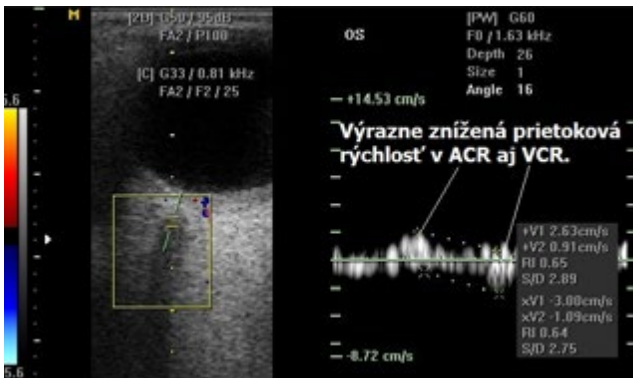

Fig. 8. Pronounced decrease of flow speed in central retinal artery upon direct carotid-cavernous fistula ipsilaterally in comparison with other eye

- **Inflammation of conjunctiva, episclera:** CCF has typical monocular hyperaemia (arterialisation of conjunctival and episcleral blood vessels), with increased intraocular pressure (Fig. 9). In comparison with typical inflammations of the conjunctiva and episclera, patients do not state burning, itching or lachrymation.
- **Endocrine orbitopathy:** "red" eye, protrusion, subjectively pressure behind the eye, ocular hypertension may occur not only in CCF but also in endocrine orbitopathy (EO). However, hyperaemia upon EO (pseudoconjunctivitis) is passive, in contrast with active arterialisation of the conjunctival vessels. Hyperaemia and tumescence of the eyelids in EO are more pronounced in early morning (or after longer period in recumbent position). Arterialisation of the conjunctival vessels is constant upon CCF. Protrusion upon EO is never pulsating, the same applies to subjective pressure behind the eyes. Ocular hypertension in EO is usually bilateral, and we measure different values of IOP in different directions of gaze – orthophoria, sursumduction and deorsumduction [23]. Upon CCF the IOP value is constant in all directions of gaze, and it is also possible to see pulsation of applanation semicircles upon applanation tonometry. Finding on B-scan ultrasonography: dilation of echo of VOS and tumescence of direct extraocular muscles [24], and above all Colour Doppler enables differentiation of CCF from EO. Likewise, if the finding does not respond to therapy for EO, it is necessary to consider CCF [25]. Vascular malformation: patients do not state any subjective retrobulbar pressure/sound. The method of choice is USD and UCD diagnosis (Fig. 10, 11).
- **Retrobulbar haemorrhage:** similarly not stated by the patient, no subjective retrobulbar pressure/sound. The method of choice is USG and UCD diagnosis.
- **Orbitopathy of malignant origin:** No pulsation, no subjective retrobulbar pressure/sound. The method of choice is NMR, CT, USG and UCD diagnosis [26].
- **Carotid occlusion:** The method of choice is UCD of the carotid system, USG / UCD of the eye and eye socket, NMR, CT examination.

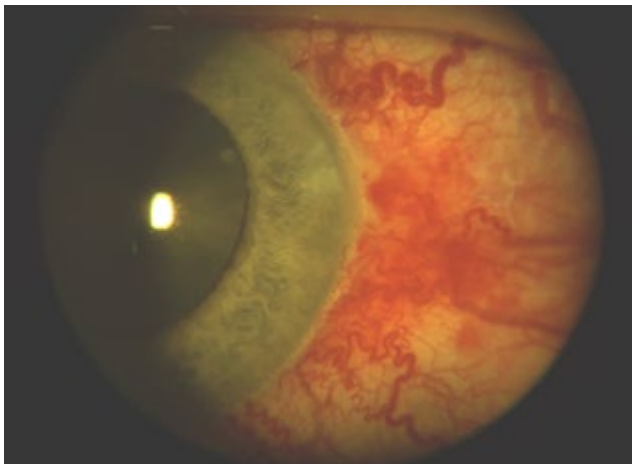

Fig. 9. Typical monocular hyperaemia (arterialisation of conjunctival and episcleral vessels) with increased intraocular pressure upon carotid-cavernous fistula in comparison with passive hyperaemia upon conjunctivitis

- **Posterior scleritis:** Although retrobulbar pain is present in scleritis, it is not pulsating synchronously with pulse. This condition is unequivocally differentiated from CCF by USG and UCD.
- **Orbitocellulitis:** CCF does not manifest pathological inflammatory markers, physiological number of leukocytes. Diagnosis is similar as for scleritis USG + UCD.
- **Thrombosis of SC:** requires radiological examination by NMR with notification of differential diagnosis of CCF and suspect thrombosis.

PREVENTION

As regards preventive measures, none exist. All that is possible is to conduct a thorough examination of all the patients who have suffered a head injury. This applies especially to those cases where the person had symptoms of skull fracture. These symptoms include periorbital haematoma, subconjunctival haemorrh-

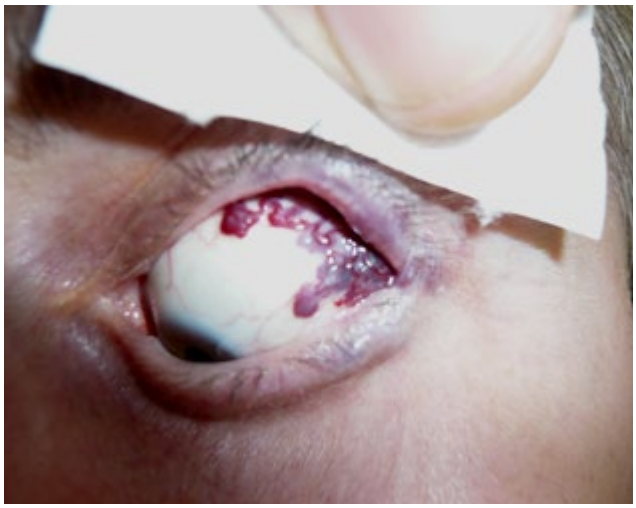

Fig. 10. Vascular malformation of conjunctival and periscleral vessels

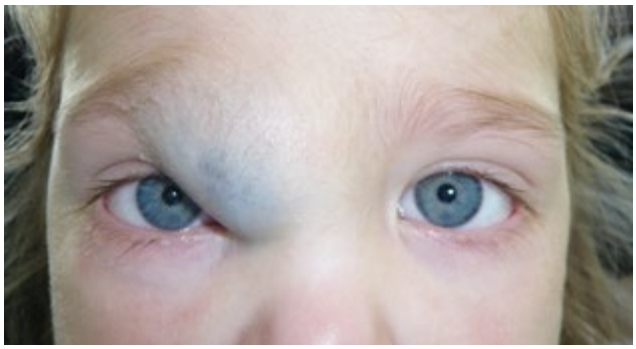

Fig. 11. Periorbital hemangioma in right eye

age, acute diplopia etc. It is necessary to diagnose CCF as soon as possible in order to ensure timely therapy and also to exclude the development of various complications.

Prognosis:

An ophthalmologist is often the first doctor to come into contact with a patient with CCF.

In the case of untreated CCF (especially direct CCF) there is a danger of severe ocular changes. According to the degree of severity of CCF, protrusion of the eyeball, chemosis of the conjunctiva, increased IOP to ischemic neuropathy of the optic nerve, central retinal vein occlusion and secondary glaucoma may occur within the course of a few days or up to several months. In addition to ocular complications, there is a large risk of intracerebral haemorrhage, pulmonary embolism, and neuropathy of the cranial nerves passing through the SC.

Approximately 20 to 30 percent of all dural fistulas lead to loss of sight, usually as a consequence of uncontrolled glaucoma, ischemic neuropathy of the optics or chorioretinal dysfunction.

Upon a diagnosis of indirect CCF, it is necessary also to examine the haemocoagulation parameters and conduct an oncological screening examination, since a clinical trial from 2017 determined a link between indirect CCFs, hypercoagulation and malignancy [27].

Following a successful medical procedure, ocular pulsation and murmur disappear within a number of hours to a few days. Dilated, congested conjunctival blood vessels, papilloedema, ocular hypertension and retinopathy usually return to the norm within the course of weeks to months. The speed and range of improvement depends on the severity of the ocular symptoms during the presence of CCF. In the case of indirect CCFs, regression is certain within 6 months at the latest. In direct CCF there may not be a complete disappearance of protrusion, ophthalmoparesis or loss of sight. With regard to the fact that dural carotid-cavernous fistulas are sometimes re-channelled following embolization or they create new abnormal vessels, it is necessary to monitor the patient and regularly observe psycho-physical functions, the ocular fundus, intraocular pressure and potentially also retrobulbar pressure by means of digital ophthalmodynamometry. However, embolisation of a CCF is linked with a number of risks which include thrombosis and reopening of the fistula. Cases have been described of worsening of angle-closure glaucoma within two months of the closure of a carotid-cavernous fistula [28]. Embolisation also increases the risk of thrombosis, especially in fragile veins [29].

THERAPY

Ophthalmological:

Treatment is standard as in the case of other causes. In the case of secondary keratopathy from protrusion of the eyeball and inability to close the eyelids, use ocular lubricants or tarsorrhaphy. More suitable is local application of medical botulotoxin to the upper eyelid. Persistent diplopia can be treated with prismatic correction, or if the eye is protruding with keratopathy, by occlusion of the eye.

In the case of pronounced ocular hypertension and secondary glaucoma, it is appropriate to choose the correct anti-glaucomatous agents. Even though the majority of cases of ocular hypertension are caused by increased episcleral venous pressure, some of them are caused by closure of the anterior chamber angle or neovascularisation of the iris. In the case of an increase of episcleral venous pressure, anti-glaucomatous agents with carboanhydrase blockers are suitable. If this does not correct the ocular condition and CCF persists, it is possible to indicate laser iridoplasty or other surgical procedures. In all cases of ocular hypertension in CCF it is appropriate to monitor also indirect signs of intracranial hypertension (ICH), or as the case may be approximate values of ICH with the aid of digital ophthalmodynamometry.

In patients with mild ocular symptoms, it is possible to wait and observe whether indirect CCF closes spontaneously, which takes place in 20–50 percent of indirect CCFs [30].

During this observation period, the patient's visual functions, IOP and ocular fundus should be monitored regularly.

In the case of ischemia and subsequent proliferative retinopathy and neovascular glaucoma, it is suitable to indicate panretinal photogocoagulation or another adequate therapy.

Conservative therapy:

As mentioned previously, the clinical course of CCF may spontaneously fluctuate or disappear completely. As a result, in the case of CCF it is also possible temporarily – before a surgical solution – to use conservative therapy. This consists in the manual compression of the ipsilateral jugular carotid artery several times per day over the course of 4–6 weeks.

In conservative therapy it is also necessary to ensure regular observation of psycho-physical functions, IOP and the ocular fundus. Higashida, Barrow et al. [11,31] state that conservative management may be effective in approxima-

tely 30 % of indirect CCFs and 17 % of direct cases of CCF.

Neuroradiological therapy:

The purpose of treatment is to close the fistula, and at the same time to maintain the through-flow of the carotid vascular system. There are a number of therapeutic options, which are being improved over the course of time. One of these is endovascular embolisation with a combination of detachable air balloons, catheters, stents or liquid embolic substances. Application is either by arterial or venous approach. It is possible to treat more than 90 % of cases successfully in this manner [32].

Stereotactic radiotherapy:

This is minimally invasive, but has a long latency period of 6–12 months: the time from performance of the procedure until closure of the fistula [33,34]. Stereotactic radiotherapy may be an option for treatment of indirect CCFs, but is not performed for direct CCFs.

Surgical procedure:

If the patient is not capable of embolisation for health reasons, or if embolisation fails, a surgical procedure comes into consideration – ligation of the internal carotid artery.

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