Polskie Towarzystwo Okulistyczne
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KLINIKA OCZNA 2022, 124, 2: 87-91
Received: 5.08.2021 Accepted: 3.10.2021

REVIEW ARTICLE

Access this article online

Website:
www.klinikaoczna.pl

DOI:
https://doi.org/10.5114/ko.2022.116829

Visual disturbances and stroke

Wojciech Lubiński[1], Barbara Nowacka[1], Lech Cyryłowski[2]

[1]2nd Department of Ophthalmology, Pomeranian Medical University, Szczecin, Poland
[2]Department of General, Dental and Surgical Radiology, Pomeranian Medical University, Szczecin, Poland

ABSTRACT

A large portion of the central nervous system is involved in visual function and therefore visual loss can be a residual manifestation of stroke. On the other hand, transient visual disturbances might be the first symptom of stroke development.

KEY WORDS: visual disturbances, cerebrovascular diseases, stroke.

INTRODUCTION

In everyday work, patients with various visual disturbances caused by cerebrovascular diseases come to the ophthalmology clinics and the emergency room. Temporary visual disturbances may be the first symptoms of an impending stroke. Rapidly introduced treatment in the stroke units of neurology clinics can prevent patients from developing severe complications after a stroke. The aim of this study is to present the current state of knowledge on the ophthalmic symptoms accompanying strokes, post-stroke complications and therapeutic options.

To understand the relationship between stroke and visual disturbances, it is important to know blood supply of the visual pathway (Figure 1) [1-6].

1. The prechiasmal optic nerve is supplied by the ophthalmic artery and internal carotid artery (ICA) pial vessels. Branch of the ophthalmic artery – the central retinal artery supplies blood to the retina.

2. The optic chiasm is supplied by the circle of Willis.

3. The retrochiasmal visual pathway

a. The optic tract is supplied by the anterior choroidal artery (ACHA) – a branch of the internal carotid artery (ICA).

b. The lateral geniculate body (LGB) is supplied by the ACHA and the lateral posterior choroidal artery (LPCHA) – a branch of the posterior cerebral artery (PCA) (dual vascular supply). The terminal anastomosis is vulnerable to ischaemia [7].

c. Optic radiations are predominantly supplied by the posterior and middle cerebral arteries and ACHA.

d. The occipital cortex is supplied by posterior cerebral arteries – the terminal branches of the basilar artery.

VISUAL CONSEQUENCES OF STROKE

Visual defects after stroke can be a cause of independence loss, depression and poorer quality of life [1, 2]. The main symptoms of post-stroke visual impairment are transient monocular visual loss (TMVL), visual field defect and ocular dysmotility.

Prechiasmal monocular visual loss might be due to retinal ischaemia secondary to ophthalmic artery occlusion [8]. Preceding symptoms often include TMVL and more than 75% of cases present a decrease in visual acuity. In 93% of patients with TMVL, ischaemia was responsible for transient ischaemic attacks (TIA) [9]. Retinal ischaemia manifests itself as amaurosis fugax or might be permanent as a consequence of the occlusion of a branch of the central retinal artery or, rarely, the ophthalmic artery. According to the American Heart Association/American Stroke Association guidelines, retinal ischaemia is included in the definition of TIA [10]. Asymptomatic patients with retinal emboli have an increased risk of stroke-related death compared with those without this retinal disease [11].

Transient binocular visual loss (TBVL) is due to the postchiasmal occlusive process in vertebral basilar arteries [12] and manifests itself as a homonymous visual field defect. This defect is a warning sign of stroke due to atrial fibrillation.

Chiasmal stroke is rare because blood is supplied through collateral circulation from the circle of Willis [13]. The most characteristic symptom of chiasmal stroke is the acute onset of bitemporal hemianopia (Figure 2). Atypical presentations can occur as transient visual loss in one eye and complete visual loss in the contralateral eye.

Postchiasmal stroke occurs due to ischaemia in the LGB, optic radiations, or occipital lobe. Visual field defects, in this

CORRESPONDING AUTHOR
Prof. Wojciech Lubiński, 2nd Department of Ophthalmology, Pomeranian Medical University, 72 Powstańców Wlkp. St., 70-111 Szczecin, Poland, e-mail: lubinski@pro.onet.pl

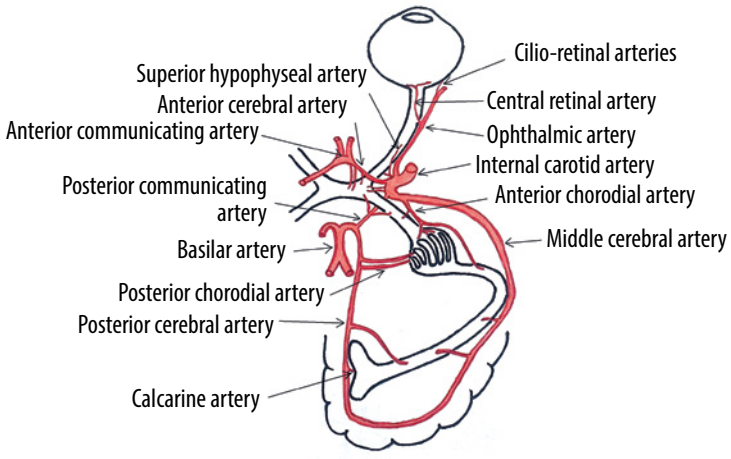

Figure 1. Blood supply to the visual pathway [1-6]

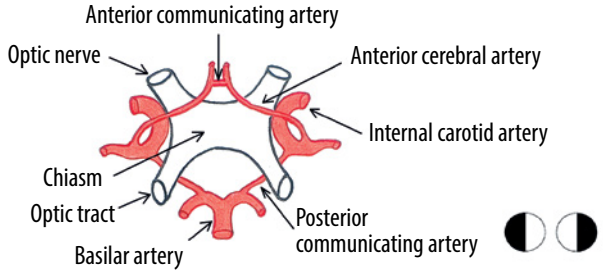

Figure 2. Optic chiasm anatomy and vasculature and an example of visual field loss in the course of chiasmal infarction.

case, are sectoranopias, quandrantanopias and hemianopias, which can be congruous or incongruous.

While contralateral homonymous hemianopia or sectoranopia might result from ischaemic injury of the optic tract and lateral geniculate body [4, 6], medial LGB infarction causes wedge-shaped contralateral homonymous hemianopia. The occurrence of bilateral LGB infarction is very rare.

What can be expected in optic radiation infarction is contralateral homonymous hemianopia or quadrantanopia. Complete homonymous hemianopia is a consequence of LPCHA and ACHA occlusion (Figure 3 and Table I) [4].

Inferior or superior quadrantanopia occurs binocularly as a result of a stroke in the occipital lobe. A stroke in this area also manifests itself as homonymous macular sparing hemianopia.

Cortical blindness is a manifestation of bilateral occipital lobe ischaemia [14]. In this case the pupillary light reflex is intact and the eye fundus is normal. If cortical blindness is connected with agnosia, Anton syndrome can be recognised [14]. An example of brain MRI with visual cortex infarction is presented in Figure 4.

Manifestation of stroke might also include changes outside the visual pathway such as ptosis, diplopia, internuclear ophthalmoplegia, one-and-a-half syndrome, gaze palsies, saccadic disturbances, smooth pursuit impairment and nystagmus.

Ipsilateral ptosis can be caused by paramedian midbrain infarction (Figure 5) [15]. Oculomotor fascicular infarction can also cause isolated unilateral ptosis without ophthalmoplegia or pupillary abnormalities (Figure 6) [15].

Acute bilateral ptosis might be due to injury of the unpaired midline central caudal nucleus. The central caudal nucleus mediates both levator palpebrae superioris muscles.

In many patients after stroke, ocular dysmotility is detected [16] and manifests itself as diplopia, internuclear ophthalmoplegia, one-and-a-half syndrome, palsies/paresis (gaze palsy, saccadic defects, smooth pursuit impairment) [17, 18]. In patients with stroke or traumatic brain injury, strabismus and third cranial nerve palsy are observed most frequently [19]. Oculomotor nerve injury symptoms are ptosis, gaze palsy, and mydriasis. Patients with stroke and associated oculomotor palsy frequently develop hemiparesis and cerebellar dysfunction. Manifestation of infarction in the midbrain might be Weber's syndrome – ipsilateral third nerve palsy and contralateral hemiparesis. Another cause of oculomotor palsy is aneurysm of the posterior communicating artery. Diplopia can be a consequence of horizontal or vertical ocular misalignment [18] after third, fourth, or sixth cranial nerve palsy. About 16.5% of cases after stroke had ocular misalignment with diplopia [16].

Horizontal conjugate gaze impairment indicates an injury of the sixth nerve nucleus involving the horizontal gaze centre [18]. Bilateral horizontal gaze palsy is rare. Internuclear ophthalmoplegia is usually unilateral and its characteristic feature is the impairment of horizontal conjugate gaze with ipsilateral adduction impairment and associated contralateral horizontal gaze-evoked abducting nystagmus (Figure 7).

Importantly, convergence is normal and this feature helps in the differential diagnosis between internuclear ophthalmoplegia and third nerve palsy. In patients over 60 years old, stroke in the medial longitudinal fasciculus is the most common cause of internuclear ophthalmoplegia [20].

Manifestation of medial longitudinal fasciculus stroke might be one-and-a-half syndrome characterized by ipsilateral horizontal gaze palsy and impaired adduction of the contralateral eye [21].

Impaired gaze holding is a result of injury to the interstitial nucleus of Cajal, nucleus prepositus hypoglossi or medial vestibular nuclei [18]. Paramedian midbrain-thalamic infarcts are responsible for complete gaze palsy, while bilateral, rostral interstitial medial longitudinal fasciculus strokes cause complete vertical palsy [22].

Voluntary horizontal saccades are rapid eye movements mediated by the frontal lobe. Cortical and brainstem infarcts can lead to saccadic defects [18].

Pursuit is the visual tracking of slower-moving targets. Impaired smooth pursuit movements can be seen in ipsilateral occipitoparietal lesions [23] and cerebellar infarctions [24].

Bidirectional horizontal nystagmus might be a manifestation of cerebellar strokes [22].

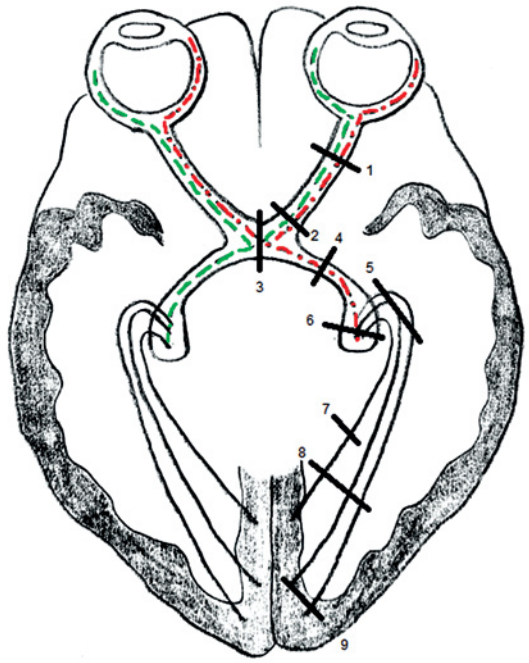

Figure 3. The relationship between the visual pathway place of damage and visual field loss

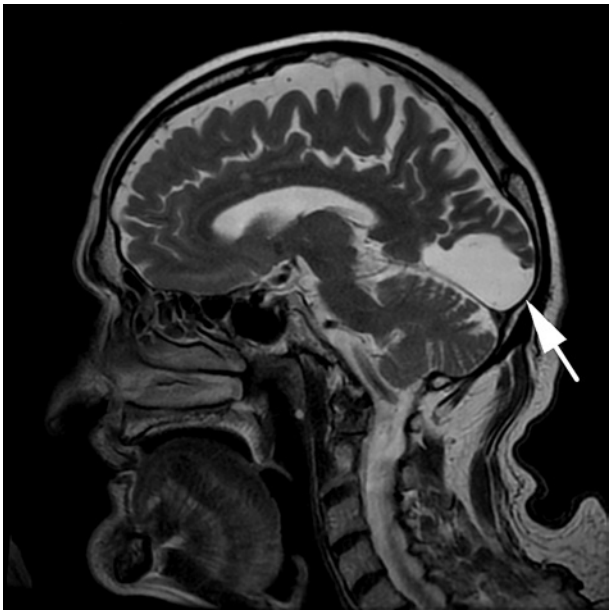

Figure 4. Sagittal T2-weighted image demonstrates encephalomalacia (arrow) following cerebral ischaemia comprising visual cortex in an 83-year-old man

In patients who have had a stroke, visual symptoms are often under-reported. It is very important to take self-reported symptoms into account because stroke can cause visual symptoms without objective, quantifiable signs of visual impairment [17]. The results of a multicentre prospective study indicated that 92% of patients who had a stroke had visual impairment even though only 42% of multidisciplinary teams reported objective findings of ocular impairment [25]. Suspected visual difficulties not reported by patients include, inter alia, neglect, closing one eye or head motion compensation.

It is suggested that retinal ischaemia is an indication for neuroimaging for stroke [10]. Stroke risk factors should be analysed, such as diabetes, hypertension, hyperlipidaemia, coronary artery disease, and smoking cigarettes. The results of a multicentre study indicated that 40% of patients with central retinal artery occlusion had ipsilateral carotid artery stenosis of 70% or more, which is why carotid Doppler ultrasonography is recommended in such cases [26].

Acute treatment for ischaemic stroke involves intravenous thrombolysis with recombinant human tissue plasminogen activator (alteplase). It converts plasminogen to plasmin, which can dissolve the thrombus and reperfuse an ischaemic brain [27]. However, the treatment should be implemented within 4.5 h of stroke onset. As the benefits decrease rapidly with increasing time after stroke onset, accurate diagnosis is critical for preventing the disability of patients [28]. Failure to recognise the symptoms of stroke is considered to be one of the main obstacles to the use of alteplase [29].

Table I. The relationship between the visual pathway place of damage and visual field loss

PLACE OF DAMAGE		VISUAL FIELD LOSS
1	The optic nerve	Monocular vision loss
2	The optic nerve at the border with the optic chiasm	Monocular vision loss with an opposite upper-temporal (junctional) scotoma
3	The optic chiasm	Bitemporal hemianopia
4	The optic tract	Contralateral homonymous hemianopia
5	The temporal part of visual radiance	Contralateral homonymous superior hemianopic defect
6	The lateral geniculate body	Contralateral homonymous hemianopia
7	The anteroparietal part of the visual radiance	Contralateral homonymous inferior hemianopic defect
8	The front part of the visual cortex	Contralateral homonymous macular sparing hemianopia
9	The posterior part of the visual cortex (responsible for macular vision)	Contralateral homonymous scotomas of central vision

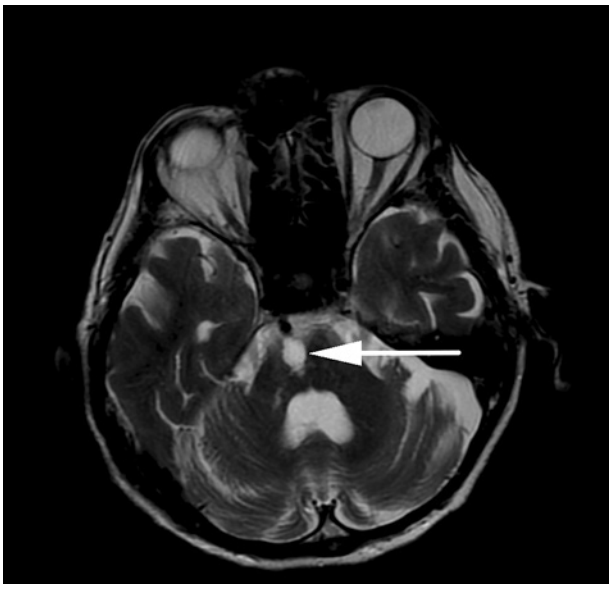

Figure 5. Axial T2-weighted (a) and FLAIR (b) images depict pontine lacunar infarct (arrow) in the area of trigeminal and abducens nerve nuclei in a 78-year-old man

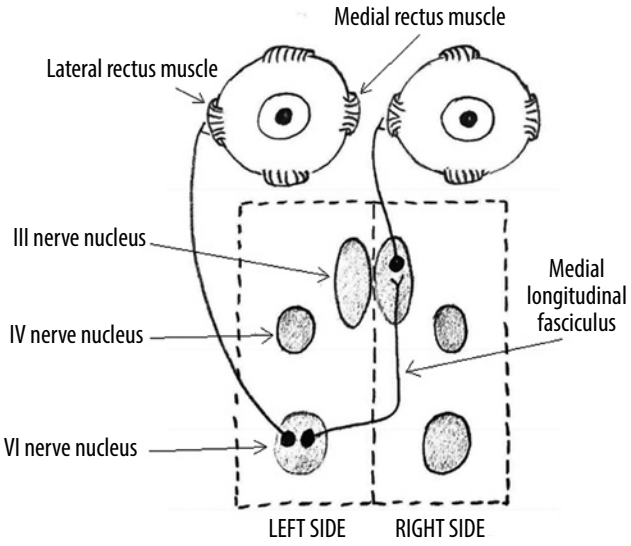

Figure 7. Anatomical pathways of horizontal eye movements. Stimulation from the nucleus of the fourth nerve runs not only to the lateral rectus muscle on the same side but also through the medial longitudinal fasciculus to the nucleus of the medial rectus muscle in the contralateral oculomotor nerve nucleus

No definitive standard treatment is available for patients with post-stroke visual field loss. Spontaneous recovery of the visual field can occur within the first 3 months after stroke [30]. Post-stroke homonymous hemianopia (HH) spontaneously resolves in 38-50% of patients [31, 32]. If the spontaneous resolution of HH is not achieved, the treatment modalities are eye movement training, optical and restorative therapy [31]. The effectiveness of vision restoration therapy depends on retrograde degeneration, i.e. stroke in the occipital lobe can lead

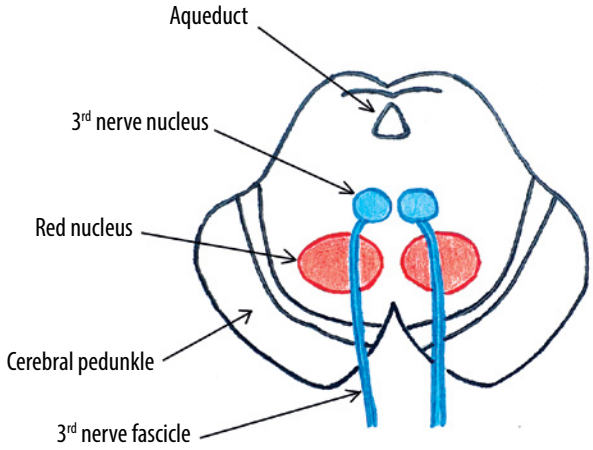

Figure 6. Anatomy of the oculomotor nerve nucleus and fascicle

to optic tract degeneration [31], which can, in turn, lead to retrograde degeneration of retinal ganglion cells.

The management of post-stroke ocular dysmotility includes convergence, saccadic and pursuit stimulation training [33], but the results are inconclusive with the exception of convergence insufficiency treatment [30]. There is evidence of the efficacy of computer-based treatment for visual field defects [30]. Substitutive devices that alter visual images, such as magnifiers, prisms or patches, might be used but the obtained results are inconclusive [33]. In the treatment of diplopia with ocular motility surgery, botulinum toxin should also be taken into account [18].

Elimination of cardiovascular risk factors, such as hypertension, lipid disorders, diabetes, smoking, abdominal obesity, various heart diseases (e.g. arrhythmias, rheumatic disease, patent foramen ovale, myxoma), vascular disease of the brain (previous TIA, ischaemic or haemorrhagic stroke), ischaemic heart disease, kidney disease, high haemoglobin concentration, reduced fibrinolysis, and oral contraceptives, plays an important role in the prevention of stroke-related vision loss, especially in patients with TIA.

Some pathological changes in the orbit or eye fundus can help in the diagnosis of cerebrovascular disease. Cholesterol plaque in the retinal artery can be a sign of thromboembolic predisposition and increased risk of future stroke. Papilloedema might be a sign of intracranial haemorrhage or brain oedema due to stroke. Vitreous and preretinal haemorrhage (Terson's syndrome) is a consequence of increased venous pressure due to increased intracranial pressure in subarachnoid or intracranial haemorrhage.

Proptosis, chemosis, conjunctival injection, tortuous vessels, and dilated ophthalmic veins are the ophthalmic signs of carotid cavernous fistulas, which are a consequence of abnormal communication between the carotid artery and cavernous sinus.

It is worth noting that in the differential diagnosis of visual disturbances caused by vascular abnormalities it is im-

portant to consider atrial fibrillation, migraine, and giant cell arteritis.

In conclusion, there are many visual symptoms of stroke. Vision abnormalities due to stroke are not widely recognised and treatment remains limited. Early, proper diagnosis of visual dysfunctions associated with stroke is very important for the patients because the fast introduction of neurological treatment provides as opportunity for the reduction of severe complications.

DISCLOSURE

The authors declare no conflict of interest.

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