BALNEO RESEARCH JOURNAL
English Edition
eISSN 2069-7619 pISSN 2069-7597 ROMANIAN ASSOCIATION OF BALNEOLOGY

Non-surgical correction of diplopia after stroke- a strong impact factor on quality of life-minireview

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Balneo Research Journal DOI: http://dx.doi.org/10.12680/balneo.2019.286 Vol.10, No.4, December 2019 p: 489–494

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Abstract
One of the most frequent disorders accompanied by diplopia is represented by stroke. Beyond different symptoms of stroke, diplopia constitutes an important clinical factor in influencing the quality of life in surviving patients. In this paper we discuss the visual impairment associated with stroke location, types of diplopia, the clinical examination, and the nonsurgical treatment of diplopia. The purpose of diplopia treatment is to restore binocular vision and eliminate the double vision. The most used nonsurgical treatment for diplopia is the optical correction by the prisms. Another therapeutic option is the botulinum toxin injections.

Key words: *diplopia, stroke, visual rehabilitation, prism correction,*

Introduction

Stroke represents the second leading cause of death (1). Commonly defined, there are two types of stroke, either ischemic (in 85% of cases) or hemorrhagic (in 15%) (1). According with the implied territory stroke is accompanied by specific clinical symptoms, which interfere with patient's quality of life (2,3).Common symptoms of stroke in the left hemisphere include aphasia, right hemiparesis and right hemianopia, and in the right hemisphere, left hemispatial neglect, left hemiparesis and left hemianopia (4). Posterior circulation or infratentorial stroke comprise a lot of additional symptoms, including diplopia, bulbar palsies, dysphagia, unilateral dysmetria and incoordination, as well as reduced levels of consciousness. Diplopia appears either in right or left-sided strokes (5).

Diplopia or double vision is one of the most unpleasant symptoms for the patient withstroke. Double vision represents the perception of two images of a single object seen adjacent to each other (horizontally, vertically, or obliquely) or overlapping. Sometimes the patients use the term of "ghost image"(6])revealed that 92% of patients who had a stroke have visual problems even though only 42% of multidisciplinary stroke team reported objective findings of ocular disorders.

Types of diplopia

Diplopia can be monocular (when the double image is recognized only by the tested eye) or binocular (when the double image is present when both eyes are open). The vertical diplopia means that one image is higher than the other and in orizontal diplopia the two images are separated horizontally.

Causes of binocular diplopia

Binocular diplopia is the most frequent form (89%) due to different pathologies including the central nervous system such as: stroke (ischemic and haemoragic), multiple sclerosis, myasthenia gravis, cerebral tumors, neuromuscular junction dysfunction, palsies of the third, fourth or sixth cranial nerves or other diseases like thyroid ophthalmopathy and diabetes mellitus (7).When one eye is closed double vision disappears (8,9,10).

Diplopia can appear as a result of different cranial nerve palsy (III,IV and VI-th)or from skew deviation (11,12) Rowe et al. (13) in a prospective multicenter observational case cohort study revealed that 16.5% of patients with stroke developed ocular misalignment associated with diplopia.

Diplopia from a recent stroke is confusing to the patient because adaptation in order to improve visual acuity by a head turn (rotational of the head to the

right/left- early stage) or suppression (capacity of the brain to ignore the information of one eye by the brain- late stage) has not yet occurred. Images may appear to overlap each other or may appear adjacent to each other. Diplopia also causes symptoms of blurred vision, dizziness, poor balance, alexia, psychological stress, asthenopia, and headaches. Sometimes patients cannot recognize double vision unless they are asked.

Double vision can be constant or variable. Binocular diplopia may vary depending on the direction of gaze or with tilting or turning of the head. Fatigue may also contribute to variable double vision.

Most stroke survivors with a known cerebral stroke-related diplopia have an overwhelming impulse to close one eye or have been instructed to patch the deviating eye in order to eliminate the second image. This makes the patient happy for the moment because the patch resolves the diplopia. Unfortunately, patching the deviating eye for too many weeks can facilitate the binocular dysfunction and reduce peripheral vision. Stroke-related binocular dysfunctions with mild-to-moderate paretic angles of strabismus are often capable of a wider range of motion of the effected eye. This can be achieved by having the patient monocularly track a moving target (pursuits) in the direction of the restrictions several times per day for a few weeks.

Severity of diplopia (mild, moderate, severe) in different gaze position may be established by the patients by completing the Diplopia Questionnaire (freelyavailableat:www.pedig.net)on a 5-pointscale (always, often, sometimes, rarely, never) (14).

Squint associated with diplopia significantly affects the patient´s quality of life. Rowe et al in his multicentre observational cohort study revealed that 16.5% of patients with stroke had squint with diplopia (13).

Diplopia can be associated with vertical gaze in palsies of the third and fourth cranial nerves (midbrain and thalamic infarcts) and with saccades of the eye mediated by the frontal lobes. The saccadic defects may appear after both cortical and brainstem infarcts (13). Another possible association of diplopia may be the bidirectional horizontal nystagmus due to a posteroinferior cerebellar strokes (15). Unfortunately visual problems after stroke are under estimated in patients with stroke (16) Hanna et al. (17) developed a screening tool for visual impairment post stroke.The authors concluded that the existing tools gave incomplete evaluation of visual troubles after stroke (17).Retinal ischemia can accompany diplopia and may act like a prelude for stroke as it stipulates the American Stroke Association guidelines, that is why neuroimaging is recommended immediately to identify a possible concomitant cerebral ischemia (18). There are studies (19) which demonstrates that 18.2% of patients with retinal ischemia developed a recurrent stroke after 1 month follow-up. A characteristic sign for thrombembolic predisposition is the Hollenhorst plaque (cholesterol plaque)in the retina (20). Papilooedema may be present due to the brain oedema secondary to stroke. (21). Posterior pole hemorrhages (Terson syndrome) as a result of intracranial hemorrhages may be present (22).

There are also risk factors which should be explored such as: diabetes mellitus, hypertension, hyperlipidemia, coronary artery disease and tobacco use which can act as a trigger for recurrences of stroke (12,23). The stroke may be associated with visual field deficits. Some authors (24,25) consider that visual field defects indicates a poor clinical outcome, risk of falls and mortality in patients with stroke.

Clinical examination

It is mandatory to follow a complete ocular exam, including the exam of visual acuity with the best correction, ocular fundus assessment, intraocular pressure measurement and refraction examination.

The presence and evaluation of diplopia starts with the ocular motility exam by testing ductions in all directions of the gaze. The motility is reduced or absent in the direction of the paretic muscle in a incomitant squint and mostly indicates a neurogenic palsy (26).

The presence or absence of eye deviation is assessed by cover/uncover, alternate cover, and/or Maddox rod testing. The amplitude of deviation can be recorded with prisms, using prism cover test and Maddox rod testing when necessary, in primary position at distance (5 m) and near (30 cm) and in all cardinal gazes.

The **prism and alternate cover test** alternates the cover over both eyes while a prism is placed in front of one eye. This helps measure the difference between the two eyes and determine what prism is needed to fix the double vision.

Fusion amplitudes is measured for patients with long-standing deviation, and presence of excyclotorsion is identified by using double Maddox rod test.

Nonsurgical treatment in diplopia

Nonsurgical treatment in diplopia includes prism prescription and botulin toxin injection.

Prism is a transparent, solid, triangular refracting medium with a base and apex. Its apical angle determines the power of prism. A prism of one prism diopter power (Δ) produces an apparent displacement of one centimeter to an object situated one meter away. Light entering the prism will deviate toward its base. However, image appears shifted to the apex, and the eye examined or treated tend to deviate toward its apex. This is how prisms function and manipulate the special location of image clinically (27).

Indications for prism correction are management of: long-lasting diplopia consecutive to cerebral stroke associated with small angles eye deviation (under 10 Δ) and transitory diplopia with or without strabismus.

When a prism is placed in front of one eye, with its base directed outward, the light rays from object will be deflected outward (to the base) and fall on the retina outer to fovea. Since the light still falls on the macula of the other eye, double vision would be produced. Consequently, the eye with prism base directed outward will deviate inward so that the deflected light falls on the macula once again and binocular vision is made possible. The maximum effort which can be put in this way (the capacity to maintain fusion) is measured by the strongest prism with which diplopia is not produced. Prismatic power of even 30– 60 Δ can be overcome normally by convergence and 10– 15 Δ divergence, and 2– 4 Δ by circumvergence. The measurements of this artificially produced diplopia (vergence power) are important in diagnosis and treatment.

There are few studies (28,29) evaluating the effects of non-surgical treatments for strabismus on the patient's life quality. Prism correction is a commonly used non-surgical treatment for binocular diplopia in the context of a variety of different strabismus types (30). Successful correction of diplopia using prism would be expected to result in significant improvements in visual function which may then translate into improved questionare of quality of life.

Prisms are generally recommended for optical correction of symptomatic binocular diplopia of different ethiologies (30,31,32,33,34). They eliminate diplopia by altering the path of light rays and aligning the image on the fovea of the deviating eye. Studies reporting data on success of prisms in treating diplopia are limited to a few case series that lack details of ocular misalignment and prism prescriptions (35,36,37,38,39). It is generally believed that prism glasses are most successful in eliminating diplopia in patients with comitant deviations of less than 10–12 prism diopters (PD), although the success of prisms for deviations greater than 10 PD and for incomitant deviations has not been systematically studied (35).

Types of prisms

If prism correction is needed for a longer period, it can be ground into the lens of eyeglasses. Eyeglasses with a prism look like any other glasses without a prism, although the lens on one side may be thicker and more noticeable.

Sometimes when we assume that the diplopia will be temporary ,or patients require larger prism strength (>10 D) and in those with greater than 5 PD of lateral incomitance, or as an initial trial to assess the effectiveness of prisms prior to prescribing permanent prisms the Fresnel prism (a thin press-on vinyl sticker) is fitted over the front of eyeglasses. With a Fresnel prism lens, the prism is slightly visible.

The advantages of Fresnel prisms are represented by the oblique application in patients with combined horizontal and vertical deviation, larger deviations (up to 30 PD) and are lightweight and thin.Moreover, they can be easily mounted to the back of the spectacle lens. The disadvantages of these prisms are the loss of contrast, loss of acuity, scattering of light, and visibility.

This is not ideal for long-term use, but it does allow the patient to test drive a prism and see how it works. Fresnel prisms are also used when the prism prescription is not stable [35].

Prescription of prisms

Like normal eyeglasses, a prism is measured and prescribed with a unit of measurement called **prism diopters** (from 0.5PD, 1.0PD, 1.5PD, and so on). Depending on the double vision, the prism is placed vertically, horizontally or diagonally in one or both lenses of the eyeglasses.

Sometimes when we assume that the diplopia will be temporary, a Fresnel prism (a thin press-on vinyl sticker) is fitted over the front of eyeglasses. With a Fresnel prism lens, the prism is slightly visible. This is not ideal for long-term use, but it does allow the patient to test drive a prism and see how it works.

Fresnel prisms are also used when the prism prescription is not stable and may change rapidly.
Prescriptions of prism cover the following steps. In general, the minimum amount of prism that would make the patient diplopia free in the primary gaze is prescribed. This is measured by moving a Barren´s prism bar with increasing strength of prisms in front of one eye with the patient fixating on the distance Snellen chart until the patient reports resolution of diplopia. As a general rule, half of the prism dioptric size as measured by the prism cover test in primary position is tried and increase or decrease based on patient's responses. Usually, the prism strength is divided equally between the 2 eyes with the base on the opposite part of deviation.
It is important to establish the prism correction for distance and near. Sometimes there are differences between the two corrections. So, the gasses for distance may correct diplopia, but they are not suitable for near (the patient may need another prismatic strength) (26).
Apers and Bierlaagh (38) described the effectiveness of prisms in 75 patients with paralytic squint. Hatt et al (28) in their study of adults with a different range of strabismus types and moderately severe diplopia, demonstrated a prism success rate of 68%.
Tamhankar et al reported success rates in adult strabismus patients.[thamkar,thamkar] For both Tamhankar et al studies success with prism was defined as complete or partial resolution of diplopia based on patient report. In the first study, of 94 patients with a range of motility disorders, 88% were considered successfully treated (12) and in the other study, of 64 patients with large angle, incomitant, or combined horizontal and vertical strabismus, 72% were successfully treated (30).There are conditions where optic corrections fail due to other ophthalmological disorders (40,41), due to associated neurological symptoms, or due to cognitive severe impairments (42).
Another treatment for diplopia is the injection of botulinum toxin which blocks local synaptic transmission at cholinergic terminals. Botulinum neurotoxin prevents the release of acetilcholine at the presynaptic neuromuscular junction thereby inducing flaccid paralysis. This toxin-mediated paralysis includes three principal steps: binding, internalization, and inhibition of the release of neurotransmitter (43).
It has become a useful therapeutic armamentarium in the treatment of post stroke spasticity. Its principal advantage is that it has a focal, selective, and reversible effect in the injected muscles without having very many adverse effects[44]Botulinum neurotoxin is significantly associated with reduced spasticity, increased range of motion, and improved upper extremity function. In the treatment of strabismus associated with stroke, it is considered that botulinum toxin causes lengthening of the injected muscle and a correlate shortening of the antagonist muscle.

Conclusions

Visual impairment is frequently under-reported in patients with stroke. Binocular diplopia with ocular deviation is a common sign in the stroke influencing the quality of life. It may be associated with other ocular sign including visual field defects. Correction of diplopia is mandatory in order to eliminate double vision. Nonsurgical treatment of diplopia includes the use of prism and botulinum toxin.

Aknowledgements

Cristina Nicula has written the majority of manuscript, performed the proofing, researched the topics and approved the final manuscript. Dorin Nicula has also written part of the manuscript as well as researching the topics .Adriana Elena Bulboaca provided a literature review and approved the final manuscript.

Competing interests: None declared.

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