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REVIEW

Post-cataract surgery diplopia: aetiology, management and prevention

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Diplopia is an infrequent but distressing adverse outcome after uncomplicated cataract surgery. Many factors may contribute to the occurrence of this problem, including prolonged sensory deprivation resulting in disruption of sensory fusion, paresis of one or more extraocular muscles, myotoxic effects of local anaesthesia, optical aberrations (for example, aniseikonia) and pre-existing disorders (for example, thyroid orbitopathy). The purpose of this review is to present the aetiology and clinical features of diplopia after cataract surgery and to discuss the possible modalities for the prevention and treatment of this frustrating complication.

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Cataracts account for a major proportion of visual loss around the world. Within the Western world cataract surgery is the most commonly performed eye procedure.[1] The standard approach consists of phacoemulsification and intraocular lens implantation under regional or topical anaesthesia as a day procedure. Expectations from cataract surgery are very high. The patient expects a significant improvement in sight with an uneventful recovery; however, like in all surgical procedures, complications can occur in cataract surgery. These complications can be divided into peri-operative (posterior capsule rupture, vitreous loss, lens material drop in the vitreous cavity, expulsive haemorrhage) and post-operative (endophthalmitis, cystoid macular oedema, iris prolapse, aqueous humour leakage from the incision site, remnant of lens material in the anterior chamber, bullous keratopathy, diplopia and retinal detachment). All of the aforementioned complications require prompt medical and/or surgical management; however, some complications (expulsive haemorrhage, endophthalmitis, retinal detachment and vesicular keratopathy) can be devastating for the patient's vision despite immediate intervention.

DIPLOPIA

Persistent diplopia is an uncommon but frustrating adverse outcome of otherwise successful cataract surgery under topical/ local anaesthetic (incidence of 0.18 per cent).[2] Diplopia can be categorised into monocular or binocular.

Monocular diplopia may arise from corneal epithelial irregularities, cataract in the contralateral eye, intraocular lens decentration, uncorrected ametropia and polycoria. Although it is more common, transient binocular diplopia has less clinical significance because it usually resolves spontaneously within a few days or weeks.[3] Usual causes of such diplopia include small, pre-existing, fusible sensory strabismus, a prolonged local anaesthetic effect and operative trauma to the orbital soft tissues.[3]

The purpose of this review is to present the aetiology and the clinical features of binocular diplopia after cataract surgery and to discuss the possible modalities for the prevention and treatment of this frustrating complication.

AETIOLOGY

The causes of persistent squint after a cataract operation are numerous and the clinical features depend on the underlying aetiology.[4] There are four main categories of patients developing post-cataract strabismus resulting in diplopia.

Pre-existing heterotropia masked by cataract

Pre-existing or concurrent disorders masked by cataract are typically from dysthyroid ophthalmopathy (DO) and demonstrate enlarged extraocular muscles on orbital imaging (computed tomography or magnetic resonance imaging).[5] Nevertheless, the diagnosis can sometimes be difficult to establish in the absence of classic signs and therefore, diplopia due to dysthyroid ophthalmopathy is generally a diagnosis of exclusion. Other causes include myasthenia gravis, ocular motor nerve palsy, childhood strabismus and amblyopia, foveal dystopia (epiretinal membrane, subretinal neovascularisation, history of macular laser photocoagulation)[6] and previous damage to orbital soft tissues (for example, buckle for retinal detachment surgery).

Sensory deprivation by the cataract

Sensory strabismus from cataract is becoming increasingly rare since opaque lenses are

removed well before vision is severely reduced in Western countries. It is still encountered in patients with long-standing unilateral traumatic cataract.[7,8] Pratt-Johnson and Tillson[7] reported 24 patients with central disruption of binocular vision following removal of unilateral traumatic cataracts, which had been present for several years. The affected eye in such patients is typically exotropic, hypotropic and cyclodeviated and we believe that it is crucial to test fusion on the major amblyoscope to correct for any torsional deviation before stipulating that central fusion is disrupted.

Another cause belonging to this category is decompensated heterophoria, such as esophoria, exophoria or long-standing superior oblique palsy that may lead to post-cataract diplopia.[9,10] Additionally, childhood strabismus or amblyopia may predispose to the emergence of diplopia after a cataract operation through:

1. elimination of suppression in an amblyopic eye with intractable diplopia[11]
2. change in strabismus angle with drift outside the suppression scotoma or
3. switching fixation to the amblyopic eye leading to fixation-switch diplopia.[12]

Optical aberrations

Optical aberrations associated with aphakia and pseudophakia include anisophoria (for example, anisometropic spectacle correction, intraocular lens decentration, uneven segment height), aniseikonia (for example, induced anisometropia in an isometrope, induced isometropia in a previous anisometrope), brightness and colour disparity between phakic and operated eye, optically induced glare and halos and also disturbance of pattern of ocular dominance (that is, fixation switch diplopia).[4,13]

Surgical or anaesthetic trauma

This last category has received much attention recently as a potentially avoidable cause of vertical diplopia. Strabismus of this type has been attributed to bridle-suture trauma[14,15] to the superior or inferior rectus muscle, direct needle trauma to muscles or related nerves from the retrobulbar needle,[16,17] ischaemic injury of the muscle from swelling and haematoma formation[18] and an inflammatory reaction to subconjunctival gentamicin;[19–21] however, most authors now believe that squint is mediated by the myotoxic effect of the local anaesthetic during the retrobulbar injection.[22–32] The capacity of commonly used local anaesthetics to permanently damage extraocular muscles is now well established. Carlson and colleagues[31] injected normal saline or various local anaesthetic agents directly into monkey extraocular muscles and found that, in contrast to the minimal damage caused in normal saline-injected muscles, anaesthetic-injected muscles showed widespread lesions. In the same study using humans, significant localised lesions were seen in rectus muscle that had been injected previously with local anaesthetic in two elderly subjects. Porter and colleagues[32] claimed that direct injection into the muscle itself may be required for significant myotoxicity to occur. Several studies[22–32] have concluded that the susceptibility to muscular injury by local anaesthetics and its degree depend on numerous factors, including the volume and concentration of the drug injected, the site of injection, the age of patients, the extent of muscle fibre regeneration, the degree of fibrotic reaction, additional injury from haematoma and damage to neural structures.

It has been described that most cases of binocular vision disorders after cataract surgery occur in left eyes.[33,34] Incidence of binocular diplopia was more frequent following cataract surgery in the left eye (73 per cent), although the difference was not statistically significant ($p = 0.075$). Authors speculate that this is attributed to the fact that most doctors are right-handed and it is technically trickier to perform peribulbar anaesthesia in the left eye, as the needle can go much closer to the muscular cone.[33,34]

Another study[35] postulates that the possibility of inferior rectus damage compared to injury of the superior rectus is 4.8 times greater in peribulbar than retrobulbar anaesthesia and also that the inferior rectus is the most frequently affected extraocular muscle.

It should not be overlooked that the inferior oblique can also be affected.[36] Four incidents of inferior oblique damage have been described in a single study. In three incidents, there was a delayed damage of the muscle and the damage was compatible to the hyperfunction of the inferior rectus, possibly due to secondary fibrosis; however, in one case, there was hypofunction immediately after surgery compatible with paralysis of the inferior oblique. The latter was possibly related to direct trauma from the injection.

Another issue that causes controversy is the use of hyaluronidase in the anaesthetic injection. There are studies where the use of hyaluronidase appears to have a protective and beneficiary role in preventing the 'myotoxicity' of the anaesthetic at a large group of patients.[37]

Similar results are demonstrated in research, where omitting hyaluronidase resulted in the appearance of many incidents of post-operative diplopia.[38–41] In a retrospective study, Brown and colleagues[41] found that hyaluronidase probably prevents damage of extraocular muscles, with the inferior rectus being the most vulnerable due to the anaesthetic technique used. Similar results appear in another study[42] concluding that hyaluronidase should be added to the local anaesthetic because of its beneficial result. A multi-centre retrospective study from the USA showed that there was a slight increase in cases with diplopia during a period of hyaluronidase shortage; however, this was not statistically significant.[2] Our personal experience coincides with the majority of the aforementioned studies, which support the use of hyaluronidase in cataract surgery for prevention of post-operative diplopia. Further prospective studies are necessary to evaluate the prevention efficacy of hyaluronidase in cataract operations.

CLINICAL FEATURES OF POST-CATARACT DIPLOPIA

Muscle injury

Periocular muscle injury following injection of local anaesthetic can affect any extraocular muscle (mainly the inferior rectus) but the levator muscle and even the orbicularis oculi muscle can also be affected, causing ptosis and lagophthalmos, respectively.[43] Muscle injury can lead to contracture, overaction or paresis.

Extraocular muscle contracture after retrobulbar anaesthesia was first described in the inferior rectus muscle.[18,28,29] Nowadays, we know that despite the fact that the inferior rectus is the most commonly affected muscle, any of the extraocular muscles can develop contracture. Specific occasions of involvement of the superior rectus, lateral rectus and inferior oblique have been described.[16,22] The commonest cause of extraocular muscle contracture is retrobulbar anaesthesia but the precise pathogenetic mechanism remains unclear. Most authors believe that the problem arises from local anaesthetic myotoxicity.[21,23] An initial paretic phase followed by

regeneration and fibrotic proliferation leads eventually to muscle contracture in most cases.[21,23]

Some patients present with overaction of an extraocular muscle. The most likely pathomechanisms are:

1. local anaesthetic myotoxicity and associated paresis of the antagonist muscle[17]
2. a botulinum toxin-like effect of the antagonist[25] and
3. initial paresis and subsequent overaction in the same muscle.[22] We must stress that overaction is closely related to contracture and the end result of muscle status depends on its response to the anaesthetic agent rather than any qualitative pathogenetic difference.

Rarely, an extraocular muscle paresis can occur, in which the inferior rectus is typically affected. Esswein and von Noorden[26] described nine patients with permanent paresis of a vertical rectus muscle (seven with the inferior rectus and two with the superior rectus) and the suggested pathomechanisms include mechanical trauma from the injection needle to the muscle's nerve supply and direct myotoxicity or nerve toxicity.

Optical aberrations

Aniseikonia is usually the first optical factor that comes to mind. Patients with long-standing, compensated aniseikonia may become symptomatic after emmetropia is achieved after cataract surgery.[4] Anisophoria is a more significant cause of diplopia than aniseikonia and it requires an anisometropia greater than 2.50 D before it becomes a problem for the patient.[13] Also, switching ocular dominance after a cataract operation may bother some patients, as some people have less tolerance to optical aberrations affecting the dominant eye.[12]

TREATMENT

Conservative management

Prisms are the mainstay of non-surgical management of post-cataract diplopia. They can be used either as a permanent solution or a temporary measure, while awaiting a squint operation. Patients with small deviations (less than 10 prism dioptres) can use either ground-in or Fresnel prisms, while individuals with large deviations find Fresnel prisms extremely helpful. An alternative to prisms is botulinum toxin injections.[44]

Surgical treatment

Prior to offering a patient an operation for squint correction, the strabismologist must verify the stability of the strabismic angle and ensure that the patient is able to fuse an image with free prisms or with the synoptophore. In most patients who suffer from extraocular muscle contracture or paresis due to surgical and anaesthetic manipulations, squint can be corrected by weakening the contracted muscle or by weakening the antagonist to a paretic muscle. Patients with extraocular muscle contracture or overaction require large recessions and adjustable sutures may be extremely helpful.[45] In general, patients with diplopia after cataract surgery are managed successfully with strabismus surgery.[3,17,25,29,46]

Patients who fail to respond well to conservative or surgical treatment are only those having a central disruption of binocular vision after removal of a long-standing unilateral traumatic cataract or those presenting with profound asymmetry in sensory input between the two eyes.[45,46]

PREVENTION

The selection of anaesthesia plays an important role in the appearance of diplopia after cataract surgery. Topical anaesthesia with eye drops (for example, proxymethicaine / oxytetracaine) or gel (for example, lignocaine) is undeniably more tempting and comfortable for the patient, as an injection is avoided; however, patient selection is crucial to ensure a safe phacoemulsification and prevent any intraoperative complications. Topical anaesthesia, has reduced significantly the possibility of post-operative diplopia.[47,48] Yanguela and colleagues[47] conducted a study with 3,542 patients, who underwent cataract surgery during a period of 2.5 years; 2,122 cataract operations were performed with peribulbar anaesthesia and 1,420 with topical anaesthesia. In the first group, there were 21 incidents of post-operative diplopia and in the second group only three. In the first group, 11 out of the 21 incidents were related to extraocular muscle injury while in the second group there were no incidents of extraocular muscle damage. The difference between the two groups was statistically significant but we note that the appearance of diplopia after a cataract operation with topical anaesthesia is very small. In case retrobulbar anaesthesia has to be administered, it is crucial that only surgeons or anaesthetists who are competent and familiar with the technique inject the local anaesthetic. Alternatively, sub-Tenon's anaesthesia with a blunt cannula should be considered. This method is an excellent substitute for retrobulbar anaesthesia, offering equivalent anaesthetic effects with few of the drawbacks associated with the retrobulbar route.[49]

In some cases, strabismus and amblyopia were pre-existing disorders, which remained unnoticed due to a dense cataract. Additionally, the presence of a dense cataract may result in the alteration of a phoria to a tropia, which post-operatively can be symptomatic.[15,38] Therefore, all cataract patients must undergo preoperative assessment of ocular motility. In cases where a more detailed examination is needed, it is useful to obtain a detailed personal and family history. Old photographs, time and place of outset, presence of compensatory head positioning, correlation to a systemic disease or injury may be useful. Additionally, all cataract patients should be informed of the possibility of post-operative diplopia and the possible need for an operation to correct the strabismus.[50]

CONCLUSION

Cataract surgery is a very common surgical procedure that apart from improving patients' vision is aiming to achieve emmetropia. Thus, phacoemulsification can be considered as refractive surgery. Persistent diplopia after cataract surgery is an uncommon but extremely frustrating complication that can significantly compromise a patient's life. The devastating effect of this post-operative diplopia can be prevented by thorough preoperative ocular motility examination, calculation of the refractive power of the fellow eye and avoidance of retrobulbar injections, especially without the use of hyaluronidase. When constant diplopia is established after a cataract operation, treatment with prisms or surgery for strabismus can eliminate the problem in the majority of the cases.[44,45]

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