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Age-related secondary glaucoma

Jaskra wtórna związana z wiekiem

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Summary

Secondary glaucoma is a disease of multifactorial etiopathogenesis. The word “secondary” indicates that its cause lies in other pathological lesions within the eye or often – outside it. One of the risk factors that predisposes to secondary glaucoma development is the patient’s age. The risk of systemic and eye diseases increases with age. Some types of secondary glaucoma may occur in patients of every age, others are more common in the group of young patients and some of them are only relevant to the group of the oldest ones. In the group of age between 20 and 40 years the most common etiopathogenesis of secondary glaucoma was trauma and an eye surgery. In the group of patients above 40 years of age the most common reason to develop the secondary glaucoma occur to be the lens pathology, cataracta surgery and the neovascularization. There are presented many types of secondary glaucoma. During the examination it is worth to consider the age of the patient as it can expedite diagnosis, appropriate treatment and avoid complicated operations with serious risk.

Key words: age, secondary glaucoma, phacolytic glaucoma, intraocular pressure, filtration angle

Streszczenie

Jaskra wtórna jest chorobą o wieloczynnikowej etiopatogenezie, wtórnej – jak wskazuje nazwa – do innych patologii leżących w obrębie narządu wzroku, a także często poza nim. Wiek pacjenta jest niewątpliwie jednym z czynników predysponujących do rozwoju jaskry wtórnej. Wraz z wiekiem wzrasta ryzyko rozwoju chorób ogólnoustrojowych i chorób oczu. Niektóre typy jaskry wtórnej mogą wystąpić u pacjentów niezależnie od wieku, inne są bardziej charakterystyczne dla młodych dorosłych, a jeszcze inne częściej spotykamy u starszych pacjentów. W grupie wiekowej do 40. roku życia najczęściej rozpoznawano jaskrę wtórna do urazów lub zabiegów chirurgicznych. Pacjenci powyżej 40. roku życia rozwijali jaskrę wtórna najczęściej w wyniku patologii soczewki lub neowaskularyzacji. Możemy wyróżnić wiele przyczyn występowania jaskry wtórnej. Powyższy podział przedstawia jedynie najczęstsze jej typy związane z wiekiem. Diagnozując chorego z jaskrą wtórna, warto więc zwrócić uwagę na jego wiek, gdyż może to niekiedy znacznie przyspieszyć diagnostykę i pozwolić na szybsze wdrożenie odpowiedniego leczenia jak również pozwoli uniknąć skomplikowanych operacji obciążonych poważnym rokowaniem.

Słowa kluczowe: wiek, jaskra wtórna, jaskra fakolityczna, ciśnienie wewnątrzgałkowe, kąta przesączania

INTRODUCTION

According to the definition by the European Glaucoma Society (EGS), the secondary glaucoma is an elevated intraocular pressure which causes progressive typical glaucomatous optic neuropathy and visual field loss caused by other ophthalmological or extraocular diseases, drugs and treatments (1). Glaucoma, both secondary and primary, is divided into two types – open-angle and closed-angle. What is more, it is possible for one form to gradually convert into the other. Another factor according to which the secondary glaucoma is classified is the cause. We can distinguish several types of glaucoma, e.g. traumatic, uveitic or lens-related. The number of causes of glaucoma makes it impossible to present all of its forms. Therefore, the main area of interest has been the relation between the

patient’s age and the occurrence of specific forms of secondary glaucoma.

In a study published by the Indian Journal of Ophthalmology, 2997 patients with suspected glaucoma were examined. The diagnosis was confirmed in 2650 cases and 579 of those patients (21.84%) were diagnosed with secondary glaucoma. The following observations were made during the analysis of the group’s age:

- patients of 0-20 years of age constituted 25%,
- patients of 21-40 years of age constituted 27%,
- patients of 41-61 years of age constituted 30%,
- patients above 60 years of age constituted 18%.

In younger patients (age below 20 years) the secondary glaucoma developed mostly due to some kind of trauma. In the group of age between 21 and 40 years, the development of glaucoma was usually

following an eye surgery. Patients of 41-60 years old usually developed secondary glaucoma during the course of neovascularization. In patients above 60 years of age, lens pathology and surgeries (mostly cataract surgery) are regarded as the main causes of secondary glaucoma (2-4).

TRAUMATIC GLAUCOMA

It is a type of secondary glaucoma which can develop at any age but statistically it is observed more often in younger, active persons without any health burdens.

Posttraumatic haemorrhages to the anterior chamber can cause an elevation of the IOP because of erythrocytes blocking the trabeculation (**erythroclastic glaucoma**). Although most of posttraumatic haemorrhages to the anterior chamber are temporary and not particularly dangerous, sometimes a prolonged and intensive IOP elevation can damage the optic nerve and cause the cornea to be infiltrated with blood. The optic nerve is at risk when an IOP of more than 50 mmHg persists for 2 days. The extent of the haemorrhage to the anterior chamber is a useful prognostic hint regarding visual acuity and risk of complications. If the haemorrhage takes less than half of the anterior chamber then in 4% of cases an IOP elevation will occur, in 22% of cases – complications and the final visual acuity will be $> 6/18$. A haemorrhage taking more than half of the anterior chamber will cause an IOP elevation in 85% of cases, complications in 78% of cases and final visual acuity $> 6/18$ in only 28% of cases.

Treatment: hospitalization. IOP lowering drugs. Miotics are to be avoided due to the possibility of pupillary block. Steroids used locally as they lower the inflammatory state and the risk of secondary haemorrhage. Mydriatics lower the risk of a new haemorrhage from the iris and the ciliary body. Surgical removal of blood when:

- IOP > 50 mmHg for 2 days or > 35 mmHg for 7 days,
- in case of early blood infiltration of the cornea as it can lead to its intensive opacification,
- when the anterior chamber is completely filled with blood (4).

Secondary angle recession glaucoma

As a result of a blunt trauma to the eyeball, the anterior part of the ciliary body can be torn (angle recession). Although traits of angle recession are observed in many patients with posttraumatic haemorrhage to the anterior chamber, after 10 years only 6-9% of them will develop glaucoma (angle recession glaucoma). Gonioscopy can show an irregular widening of the ciliary body which can be accompanied by cicatrization and pigment in the area of angle recession. In a cursory examination, angle hyperpigmentation can be erroneously diagnosed as pigmentary glaucoma.

Treatment: The most effective treatment is trabeculectomy with antimetabolites. An artificial filtration fistula can be applied in case of trabeculectomy failure (4).

Ghost cell glaucoma

The ghost cell glaucoma is caused by blocking of the trabecular meshwork by lixiviated erythrocytes which stem from the blood extravasated to the vitreous humour. After losing their haemoglobin, empty, inflexible erythrocytes penetrate the damaged limited membrane of the vitreous humour to the anterior chamber where they settle on the trabecular meshwork. This occurs ca. 3 months after the trauma with haemorrhage in an aphakial eye and sometimes after vitrectomy and cataract removal. A suspension of green-brown coloured cells, visible with slit lamp, appears in the anterior chamber.

Treatment: IOP-lowering drugs, anterior chamber irrigation, vitrectomy in exceptional cases (4).

In patients between 21 and 40 years of age, as mentioned above, the most frequently encountered is a surgery-related glaucoma. An increase in IOP can be observed immediately after the surgery as well as some time after it. There are several causes which can be the reason for IOP increase, namely: pigment dispersion (pigmentary glaucoma), haemorrhage, ghost cell, inflammations, viscoelastic, epithelial ingrowth (1).

PIGMENTARY GLAUCOMA

The IOP elevation is currently thought to be caused by the intratrabecular spaces being clogged by the pigment and by damage to the trabeculation which is secondary to its lowering, collapse and hardening. **Pigment dispersion syndrome (PDS) is considered to be the leading cause of its occurrence.** It is characterised by a release of pigment granules from the pigment epithelium of the iris and their accumulation in the anterior segment of the eye. The release of pigment is caused by mechanical rubbing of the posterior pigment layer of the iris against small ligaments of the ciliary zonule as a result of an excessive bulging of the iris in its middle area. A possibility exists of certain abnormalities in the very pigment epithelium of the iris which facilitate the release of the pigment molecules. In some people an overly strenuous effort can cause episodes of pigment dispersion accompanied by IOP elevation. In the course of pigment dispersion syndrome, glaucoma usually manifests in 1/3 of patients in aged 30-40 years. Pigment granules are released into the ventricular fluid, move with its current and accumulate in all structures of the anterior chamber including ciliary zonule fibres. A cataract surgery can lead to a release of the pigment and to trabeculation blocking, which causes IOP elevation. The symptoms are similar to glaucoma in the course of PDS: IOP elevation, Krukenberg's spindle – vertical pigment accumulation on the corneal endothelium, small pigment granules on the iris and lens, iris pigment epithelium atrophy which leads to images of slit-like, radial defects which cause a transillumination effect, and trabeculation hyperpigmentation in gonioscopy.

Treatment: IOP-lowering drugs, laser trabeculoplasty (effects are better in younger patients), laser iridotomy, trabeculectomy (2).

INFLAMMATORY GLAUCOMA

Inflammation and haemorrhage are a frequent consequence of surgical operations. If they occur, they can lead to fibrosis and anatomic changes. Fibrosis is more commonly found in certain patients (e.g. diabetes patients or with history of uveitis), and after using certain types of lenses, such as anterior chamber intraocular lens. The inflammatory glaucoma can develop as a result of various mechanisms such as oedema of the filtration angle structures, dysfunction of endothelial cells in the angle area, trabeculation block by inflammatory cells and fibrin, breakdown of the blood-aqueous humour barrier resulting from prostaglandins activity or by steroid-induced reduction in aqueous humour flow through trabeculation (3-6). Secondary glaucoma is especially common in Fuchs' iritis syndrome and in chronic inflammation of the anterior segment of the vascular membrane. Most of the cases of anterior inflammation of the vascular membrane can be classified as idiopathic. Presence of corneal precipitates and a constricted pupil suggest that iritis is the cause of elevated IOP. During a gonioscopic examination, delicate sediments in the filtration angle can be observed. Sometimes peripheral anterior and posterior synechiae with iris bombe can form and lead to angle closure.

The treatment of glaucoma is made more difficult by several factors: corticosteroid therapy can lead to IOP elevation through minimising of the inflammatory process, by decreasing of the inflammatory and an improvement in aqueous humour production or by limiting of the outflow. Miotics are to be avoided in patients with iritis as they can intensify the inflammatory process and lead to posterior synechiae. In the case of active inflammation, IOP elevation should be ascribed to the inflammatory rather than to state steroids application (2, 4, 5). Surgical treatment includes trabeculectomy, artificial filtration fistula, cyclodestruction (2).

Viscoelastics are a technologically advanced biopolymers used during operations to protect the endothelium and maintain the intraocular space. Two basic types can be distinguished – cohesive and dispersive. Cohesive viscoelastics are usually responsible for the early postoperative IOP increase. Remains of the viscoelastic can sometimes accumulate in the trabeculation. Washing out the substance depends on its type and on the patient's clinical state. Usually, in 16 hours after the operation, the substance should be absorbed and the IOP should normalize (2).

EPITHELIAL INGROWTH

Epithelial ingrowth is a rare complication after an anterior segment or trauma operation and may potentially lead to blindness. Corneal or conjunctival epithelium cells migrate through the wound and proliferate

in the area of the anterior segment in a diffused form or by forming cysts. The IOP increase is caused by overlapping of several mechanisms, such as presence of peripheral anterior synechiae, often already existing, destruction of trabeculation by epithelial membrane and blocking of the trabeculation by desquamated epithelial cells and accompanying inflammatory cells. The diagnosis consists of identifying a persistent post-operative anterior uveitis, a diffused proliferation of the epithelium in a form of transparent membrane of undulant borders on the surface of the corneal endothelium and on the anterior surface of the vitreous humour in the area of incision and also identifying pupil deformations. The essence of the treatment is an eradication of the ingrowing epithelium to avoid a relapse. A wedge resection or a transscleral cryotherapy can be applied. The artificial filtration fistula is used when conservative therapy does not yield results (2).

In patients older than 40 years, neovascularization is considered to be the most common cause of glaucoma. It is an often occurring and severe type of glaucoma, caused by various diseases characterised by hypoxia of the retina or of the entire eyeball, or by eyeball inflammation. The most common causes are diabetes, central retinal vein occlusion or internal carotid artery stenosis. Diabetes occurs in 1/3 of neovascular glaucoma patients. The probability of NVG (neovascular glaucoma) is much higher in patients diagnosed with proliferative retinopathy. Diabetes patients who underwent vitrectomy are also at a higher risk of developing NVG. Rupture of the posterior capsule resulting from cataract surgery complications or even after using Nd:YAG laser during capsulotomy can cause VEGF to access the anterior segment more easily and accelerate neovascularization (1, 4). Ischaemic thrombosis of the central retinal vein is the cause of NVG in 36% of cases. The NVG develops in 50% of eyes with ischaemic thrombus. Glaucoma usually develops in 3 months after the thrombosis ('100 day glaucoma') (2). Internal carotid artery stenosis is the third most common cause of NVG and can easily be overlooked due to unusual symptoms. Quite frequently an IOP decrease can occur despite the neovascularization in the angle and in the iris. This is caused by a decreased ciliary body perfusion which manifests as a lowered production of aqueous humour. Early diagnosis of significant carotid artery stenosis is of utmost importance as it can save the patient's eye and sometimes even life (7). Independently from the partial overlapping of symptoms, it is worth dividing the NVG into three stages: iris neovascularization, primary open-angle glaucoma and secondary closed angle glaucoma caused by synechia (2).

Neovascularization of the iris (NVI) usually starts as small vascular patches on the pupillary margin. As the vessel grows, these patches elongate radially on the iris and take over the filtration angle (neovascularization of the angle – NVA). In the angle, new vessels branch off and form a fibrovascular membrane which

blocks the trabeculation causing secondary open-angle glaucoma. In some cases, NVA can precede the NVI. This can take place in, for example, central retinal vein thrombosis. Because of that, an accurate gonioscopic examination is crucial. An angiography of the iris and filtration angle can confirm the presence of clinically visible NVI and NVA as well as recognize their subclinical forms. However, such examination is expensive and thus not commonly available, unlike gonioscopy which, when performed accurately, can detect neovascularization just as early (7).

The therapy consists of IOP reduction and causal treatment. Panretinal laser photocoagulation (PRP) is a method of choice in all cases. The PRP can stabilise the IOP in primary open-angle glaucoma and can enhance the effects of filtration procedures if it is used in the preoperative period. The mechanism of PRP is not entirely clear. Most probably the partial destruction of the retinal pigment epithelium, which is responsible for most of the oxygen usage by the retina, enables the oxygen to penetrate from the choroid to the more inner layers of the retina and thus lowering the production of vascular growth factor. It has been reported that at least 1200 to 1600 laser foci causes NVI regression of much higher degree than their lesser number (7).

Treatment: lowering the IOP; drugs – prostaglandins are not effective due to their mechanism of action, β blockers and systemic drugs are preferred. However, very often it is not possible to reach a stable lowering of the pressure. In such cases, a surgical procedure is necessary. Anti-inflammatory treatment: steroids in drops and cycloplegics (atropine) – they lessen the inflammatory reaction as well as pain (2, 4).

One of the most common ophthalmologic diseases, which occurrence increases as the body ages, is cataract. The changes taking place in the ageing lens are often the cause of IOP elevation. Most of the glaucomas of this type can be associated with symptoms of inflammation in the eyeball. Therefore, apart from the IOP decrease, sometimes with systemic drugs, it may be necessary to treat the inflammatory process.

PHACOLYTIC GLAUCOMA

Phacolytic glaucoma (lens protein glaucoma) is an open-angle glaucoma which occurs in eyes with mature or hypermature cataract and typically in elderly people. It appears more frequently in developing countries where cataract patients do not contact their doctors immediately. Trabeculation plugging is caused by multimolecular lens proteins, which penetrate through the undamaged crystalline capsule to the aqueous humour, and also by macrophages burdened with lens proteins.

The early stage of the disease manifests as pain. Visual acuity is already low due to cataract. Examination in slit lamp shows a corneal oedema, hypermature cataract and deep anterior chamber. White particles can be floating in the aqueous humour and with enough density, they can form a pus level (pseudohypopyon).

Treatment: after stabilising the IOP and the inflammatory process with conservative therapy, the cataract should be removed and the anterior chamber should be rinsed out of the protein material. Additional care should be taken during anterior capsulotomy to avoid rupturing of the lens ligamentous apparatus (2). The prognosis depends on the duration of the elevated IOP and the degree of damage to the optic nerve. In one of the examinations, patients above 60 years of age in whom the elevated IOP lasted for more than 5 days despite the surgical procedure, had more unfavourable prognosis than younger patients with shorter duration of the symptoms (5, 8-17).

PHAKOMORPHIC GLAUCOMA

Phacomorphic glaucoma is a secondary acute closed-angle glaucoma caused by intumescent cataract. Vision deterioration or increased myopia in medical history. The lens grows throughout the whole life. Enlargement of the lens in the equator loosens the ciliary zonule ligaments and causes the lens to move forward, and with an anteroposterior diameter increase it increases iris-lens adherence which favours pupillary block.

The symptoms resemble a primary acute angle closure with shallow anterior chamber and dilated pupil. Cataract is rather evident. An examination of the second eye can show a deep anterior chamber and an open angle, which usually excludes the primary angle closure.

Treatment: laser iridotomy performed after IOP stabilisation. The cataract is removed when the eye is no longer irritated (2).

PHAKOANAPHYLACTIC GLAUCOMA

Phakoanaphylaxis is a rare state in which a patient becomes allergic to their own lens proteins which are released as a result of a surgical procedure or trauma which leads to granulomatous inflammation. The clinical picture is quite varied but in most patients a moderate inflammatory reaction in the anterior chamber occurs, as well as presence of sediments on the corneal endothelium and on the front surface of the lens. Additionally, a small inflammatory reaction in the vitreous, formation of synechiae and lens debris in the anterior chamber can be observed.

The treatment is based on using corticosteroids and aqueous humour production reducing drugs. If the pharmacological treatment proves ineffective, the lens debris should be removed (2).

SECONDARY GLAUCOMA IN THE COURSE OF PSEUDOEXFOLIATION SYNDROME (PEX)

The pseudoexfoliation syndrome is a disorder in which a grey-white, fibrous material of the extracellular matrix, produced by the ageing trabeculation epithelium cells, crystalline capsule equatorial part cells, iris and ciliary body cells, accumulates on the front surface of the crystalline capsule, on

ligaments, ciliary body, iris, trabeculation, front surface of the aqueous humour and on conjunctiva.

It is the most common cause of secondary glaucoma. In the United States, PEX have been observed in 0.6% of persons aged 52-64 years and in persons aged 75-85 years it occurred in as many as 5%. The secondary glaucoma in the course of pseudoexfoliation syndrome, unlike other types of glaucoma, poorly responds to conservative treatment and can quickly lead to severe damage to the optic nerve. PEX rarely occurs in persons of less than 50 years of age. Diagnosis of the pseudoexfoliation syndrome can be performed successfully with a slit lamp. The examination is sensitive in 85% and specific in almost 100%. Cornea – PEX on endothelium, anterior chamber – sometimes a slight clouding of the ventricular fluid, iris – PEX on pupillary margin, sphincter atrophy (defects looking as though 'eaten out by clothes moths'), front surface of the lens – central disc and peripheral band with a clear zone between it, trabeculation – hyperpigmentation resembling 'dandruff', poorly dilating pupil (18). Diagnosis: IOP measurement, slit lamp examination, gonioscopy.

Treatment: there are several methods of treating secondary glaucoma in the course of pseudoexfoliation syndrome. The method should be chosen individually for every patient. Traditional pressure-lowering drugs are not as effective in glaucoma in the course of PEX as they are in POAG but are often used as first-line therapy. These are mainly β -blockers, selective alpha receptor agonists, inhibitors of carbonic anhydrase and prostaglandin analogue. The second-line therapy is either argon laser treatment (ALT) or selective laser trabeculoplasty (SLT). Most studies show a good response to ALT and SLT. If drugs and laser therapy are insufficient for proper IOP control, trabeculectomy should be considered.

Patients with the secondary glaucoma in the course of pseudoexfoliation syndrome are at a higher risk of developing cataract and are more likely to suffer complications during opacified lens removal. Poorly dilating pupil along with a weakened ciliary zonule make the cataract surgery technically difficult. Postoperative symptoms such as inflammation, opacification, shrinkage of the posterior capsule or artificial lens displacement also occur more often. It seems that central retinal vein thrombosis can be connected with the occurrence of PEX. All this causes the secondary glaucoma in the course of pseudoexfoliation syndrome to lead to much worse prognoses than primary open-angle glaucoma.

Current research on pseudoexfoliation syndrome are aimed at establishing the genetic factors which can be responsible for the syndrome's occurrence. In numerous studies both the autosomal dominant inheritance and mitochondrial factor have been described. Most probably, however, PEX is a late-onset, multigenically inherited disorder (18).

If the symptoms appear in one eye, it is important to examine and observe the other eye very carefully. The secondary glaucoma in the course of pseudoexfoliation syndrome will most probably develop in the other eye in 40% of cases (18).

CONCLUSIONS

We can distinguish many causes of the secondary glaucoma. The aforementioned division presents only the most common, age-related types of glaucoma. When examining the patient, it should be remembered that early diagnosis, especially in the case of secondary glaucoma, is crucial as treatment of advanced secondary glaucoma is extremely complex and surgical operations necessary are often burdened with unfavourable prognosis.

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