Review

# Pseudoexfoliation Glaucoma: Clinical Presentation and Therapeutic Options

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## Abstract

Pseudoexfoliation syndrome (PES) is one of the most common causes of open-angle glaucoma, with a higher risk of vision loss, a higher maximum and mean intraocular pressure (IOP) at diagnosis, and a wider range of IOP fluctuation compared to primary open-angle glaucoma. Patients with this syndrome have a ten-fold higher risk of developing glaucoma than the normal population. A definite diagnosis can be made by the observation of pseudoexfoliation material (PEM) on the anterior lens surface, ciliary processes, zonules, and iris. PEM deposits on the zonules may explain the clinically observed zonular weakness and lens subluxation or dislocation. An increased incidence of cataract development is also associated with PES. There is growing evidence for systemic associations of PES with peripheral, cardiovascular, and cerebrovascular system diseases, Alzheimer's disease, hearing loss, and increased plasma homocysteine levels. Indications for surgery are markedly more common in patients with pseudoexfoliation glaucoma than primary open-angle glaucoma. The goal of this article is to review the latest perspectives on the clinical features, therapy, and systemic associations of this clinically and biologically challenging disease.

**Keywords:** Pseudoexfoliation, pseudoexfoliation syndrome, pseudoexfoliation glaucoma

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## Introduction

Pseudoexfoliation syndrome (PES) is the most common identified cause of open-angle glaucoma. Pseudoexfoliation is an independent risk factor for open-angle glaucoma in every country around the world. It is important to distinguish pseudoexfoliation glaucoma (PEG) from primary open-angle glaucoma (POAG). PEG is a severe type of glaucoma with a higher risk of vision loss, higher maximum and mean intraocular pressure (IOP) at diagnosis, and wider range of IOP fluctuation compared to POAG.[1]

PEG may cause increased outflow resistance as a result of progressive accumulation of pseudoexfoliation material (PEM) in the trabecular meshwork, which contributes to alteration of retrobulbar blood flow and optic nerve microvascular blood flow, as well as elastosis of the lamina cribrosa[1].

A definite diagnosis can be made by observing PEM on the anterior lens surface. These deposits can be found initially on the ciliary processes, zonules, and iris. Deposits of pseudoexfoliation on the zonules may explain the clinically observed zonular weakness and lens subluxation or dislocation. An increased incidence of cataract development is also associated with PES[1].

PES may be an important marker for cardiovascular and cerebrovascular diseases.[2] There is growing evidence for systemic associations of PES with peripheral, cardiovascular, and cerebrovascular system diseases, Alzheimer's disease, hearing loss, and increased plasma homocysteine levels.[3,4]

Clinical experiences show that controlling IOP is more difficult in PEG than in POAG. PEG patients with high IOP and advanced damage may benefit from a combination of anti-glaucoma medications as initial therapy. The indications for surgery are markedly more common in patients with PEG than in POAG.

The purpose of this article is to review current perspectives on the clinical features, therapy, and systemic associations of this clinically and biologically challenging disease.

**Background**

Pseudoexfoliation is a late-onset, stress-induced elastotic disorder that causes an aberrant extracellular fibrillar matrix material to accumulate in ocular tissues. PEG is glaucomatous optic neuropathy and IOP elevation associated with PES.

Pseudoexfoliation was first described by Lindberg in 1917 as grayish flecks on the pupillary border accompanied by chronic glaucoma.[5] The source of this material was proposed by Vogt,[6] who termed it "senile exfoliation" and suggested an origin from the lens. In 1954, the term "pseudoexfoliation" was suggested by Dvorak-Theobald[7] to distinguish the disease from the exfoliation seen in glassblowers.

**Epidemiology**

There has been increasing interest in epidemiological studies focused on PES.[16]According to these studies, this condition may be seen in between 10% and 20% of people in the general population above the age of 60. An estimated 70 million people may have PES worldwide. This syndrome occurs more frequently in Finland, Scandinavia, Greece, and Türkiye than in other countries. The differences in the rates are attributed to genetic and environmental or unknown factors. The worldwide prevalence of PES is increasing. Many patients with this condition could be undiagnosed.

PES is age-dependent, and the prevalence increases with age. Pseudoexfoliation affects both sexes, but there are conflicting results concerning its sex distribution. Some studies have found equal numbers among men and women.[8] Others have found a greater prevalence in females, while other investigators have found a greater prevalence in males.[9,10,11]

PES is one of the most prevalent causes of open-angle glaucoma, and patients with this syndrome have a ten-fold higher risk of developing glaucoma than the normal population. Pseudoexfoliation is an independent risk factor for open-angle glaucoma in every country, although reported prevalence rates vary. Population studies have suggested an incidence between 20% and 60% of all open-angle glaucoma. This syndrome is not only a cause of glaucoma but also a risk factor for glaucoma progression.

PES is often a bilateral, asymmetric disease. Both eyes may be involved histopathologically, while the clinical presentation may be seen in only one eye.[12,13,14]

As a result, PES and glaucoma are regarded as public health problems in older age.

**Genetics**

PEG is a complex genetic disease. Both genetic and non-genetic variables have a role in the etiopathogenesis.[15,16,17] The *lysyl oxidase like 1* (LOXL1) gene has been discovered as a significant genetic risk factor for both PES and PEG.[18] Lysyl oxidases are essential for the synthesis and stability of elastic fibers. Increasing evidence suggests that *LOXL1* is markedly dysregulated depending on the stage of fibrosis. In the early phases of pseudoexfoliation, *LOXL1* is involved in the synthesis and aggregation of pseudoexfoliation fiber deposits, and in the advanced stages may affect elastin metabolism. As a result, it has been suggested that pseudoexfoliation is a type of elastosis caused by a high amount of elastic microfibrillar materials like fibrillin.[19] Other candidate genes have also been reported. *CACNA1A*, *POMP*, and *SEMA6A* variants have been linked to extracellular matrix metabolism, ubiquitin-proteasome system, calcium signaling, and lipid biosynthesis in pseudoexfoliation pathogenesis, increasing the disease risk.[20]

Other non-genetic factors related to pseudoexfoliation, including oxidative stress and low-grade inflammation, can influence the expression of *LOXL1*.[16,17] A detailed study of the gene maps of this complicated disease, as well as the functional effects and molecular mechanisms of these loci, will shed light on the disease's pathophysiology.

**Clinical Manifestations**

It is important to emphasize that PES is a significant ocular problem. Most patients with pseudoexfoliation are asymptomatic.

Older patients should be carefully examined for diagnosis of pseudoexfoliation by biomicroscopy. Pupillary dilation is necessary to detect deposits on the lens surface. Classic signs of PES include fluffy, white deposits at the anterior lens surface and pupillary margins.

Pseudoexfoliation may also appear on the zonular fibers, ciliary processes, corneal endothelium, trabecular meshwork, intraocular lens, and anterior vitreous face in cases of aphakia.[13,21,22]

The bush-like fibrillar PEM may be observed in light microscopy, and the electron microscopic presentation of PEM in ocular and extraocular tissues has been demonstrated by Schlötzer-Schrehardt.[23,24]

Ultrasound-biomicroscopy studies on morphological changes in the anterior segment of eyes with PES revealed zonular weakness, thickened lens, narrow anterior chamber, and occludable angles.[25,26] Similar morphological changes in affected and fellow eyes were observed in another study comparing the involved and non-involved fellow eyes of PES patients.[27] A study using anterior-segment optical coherence tomography on patients with unilateral PES found that eyes with PES had a narrower anterior chamber angle, reduced angle widening during pupil motions, and greater iridolenticular contact and iris curve compared to healthy subjects' eyes.[28] Furthermore, non-involved fellow eyes presented similar characteristics to some extent.[27]

**Intraocular pressure**

Eyes with PES were shown to have higher IOP than non-involved fellow eyes.[11] This difference is approximately 2 mmHg. Diurnal IOP fluctuation is also greater in patients with PES than in non-pseudoexfoliation subjects.[29]

IOP may increase after pharmacologic dilation. Eyes with pseudoexfoliation should be measured after dilation, particularly due to significant pigment release.[30] Initial IOP is the strongest risk factor for developing PEG.[31,32]

**Tear film**

PEM is associated with reduction in tear secretion and tear film stability.[33,34,35] A study demonstrated that tear osmolarity in

both eyes of clinically unilateral PES patients is higher compared to normal subjects.[36]

Schirmer and tear film break-up time test results were decreased in PES and PEG groups compared to healthy controls.[36] Additionally, unilateral pseudoexfoliation was found to be associated with significant loss of meibomian gland area and higher meiboscores in both eyes.[37]

### Cornea

Small, fluffy, white pseudoexfoliation deposits may be observed on the corneal endothelium in patients with pseudoexfoliation (Figure 1) together with some pigment deposition on the central corneal endothelium (Figure 2).

Corneal endothelial cells may show changes in number and morphology. Patients with pseudoexfoliation have reduced basal epithelial and endothelial cell densities.[21,38] The damaged corneal endothelium in eyes with pseudoexfoliation can result in endothelial decompensation.[1,24,39] Endothelial cell polymegathism and pleomorphism in pseudoexfoliation keratopathy with glaucoma is more frequent than with cataract.[40] Corneal sensitivity is markedly lower in eyes with pseudoexfoliation and is correlated with the decreased basal epithelial cell and subbasal nerve densities.[41] Corneal thickness varies.

### Iris

Deposits of PEM on the pupillary border and stroma and muscle tissues of the iris are among the changes seen anterior to the lens (Figure 3). Pseudoexfoliation is associated with pigment loss from the pigment epithelium over the iris sphincter, loss of pupillary ruff, and transillumination defect of the pupillary border.[22] The iris appears to be more rigid and often dilates poorly. Iris blood vessels may become damaged and obliterated, leading to hypoperfusion of the iris. In the late stages of pseudoexfoliation, vessel wall cells may be totally destroyed.[1] Reduced oxygen in the anterior chamber is an important consequence of iris vasculopathy and chronic blood-aqueous barrier breakdown in PES. Posterior synechia may develop and contribute to insufficient pupil dilation (Figure 4).[24]

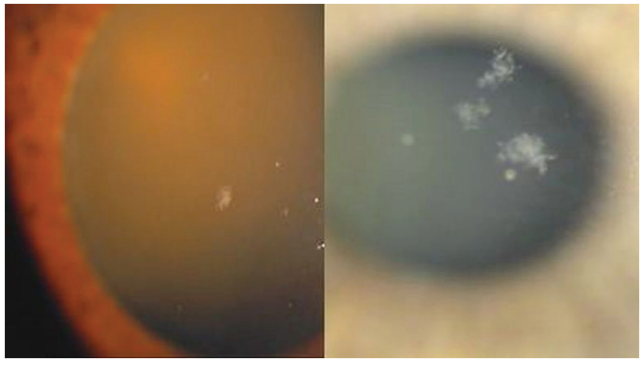

**Figure 1.** Pseudoexfoliation material deposition on the corneal endothelial surface in pseudoexfoliation syndrome

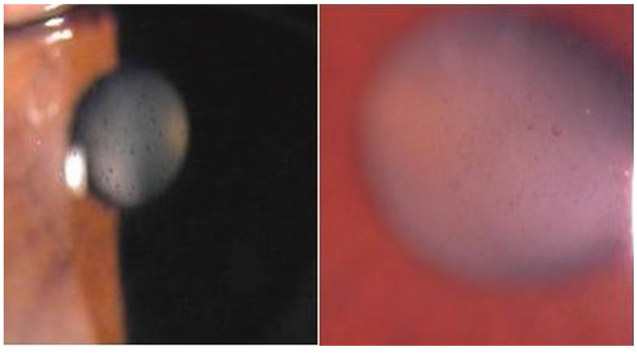

**Figure 2.** Pigmentary deposits on the corneal endothelial surface in pseudoexfoliation

### Lens

PES can be diagnosed by the observation of deposits of white material on the anterior lens surface. The epicapsular deposition appears as a homogenous diffuse ground-glass or matte film on the lens surface. As the epicapsular layer thickens, focal defects occur in the mid-peripheral zone. The classic appearance consists of a central disk, peripheral zone, and clear intermediate area. Eventually, pseudoexfoliation deposition with various appearances can be observed on the anterior lens surface (Figure 5). PEM can also be found on the surface of an implanted posterior chamber intraocular lens and the hyaloid face.[1,24,39]

### Anterior chamber angle

The defining gonioscopic feature of PES is increased trabecular meshwork pigmentation, which often manifests as patchy involvement.[42] The pigmentation is more prominent inferiorly. It is not as dense as that seen in pigmentary glaucoma (Figure 6). Small dust-like white pseudoexfoliation deposits may be observed at the angle.

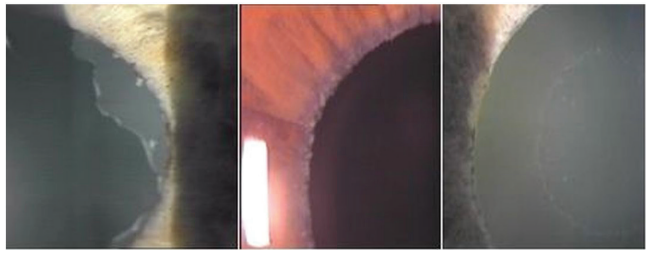

**Figure 3.** White deposits or flakes and "moth-eaten" pattern on the pupillary margin

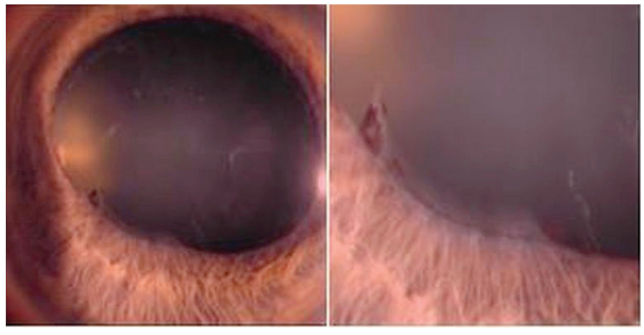

**Figure 4.** Pigment loss from the peripupillary pigment epithelium of the iris and synechiae between the pupillary border of the iris and anterior lens surface

In patients with pseudoexfoliation, gonioscopically determined angle pigmentation correlates more significantly with a higher presenting IOP than with the quantity of PEM on the anterior lens capsule.[42] The involved eye may have a narrower angle than the non-involved fellow eye.[28]

### Zonules

Small dots and flakes of pseudoexfoliation deposits can be found earliest on the ciliary processes and zonules. Deposits on zonules may explain the clinically observed zonular weakness and lens subluxation or dislocation (Figure 7).[39] Deposition of PEM on the zonules can be determined by high-resolution ultrasound biomicroscopic examination.

## Ocular Associations

### Cataract

Progressive opacification of the lens is associated with PES (Figure 8).[43] Nuclear sclerosis is the most frequently seen cataract with pseudoexfoliation. Cataract development can be explained by a setting of ocular ischemia, elevated growth factor levels, or reduced ascorbic acid levels in the aqueous humor.[24,44]

Cataract surgery in these patients predisposes to intraoperative and postoperative complications including posterior capsule rupture, zonular rupture, vitreous loss, increased postoperative inflammation, anterior capsule contraction, need for increased secondary intraocular lens implantation, and increased posterior capsule opacification.[45,46,47] However, recent advancements in cataract surgery techniques and instruments have substantially enhanced operative handling of patients with pseudoexfoliation. Final success rates for PES patients undergoing cataract surgery may even be comparable to those for non-PES patients with current preoperative, intraoperative, and postoperative techniques.[48,49]

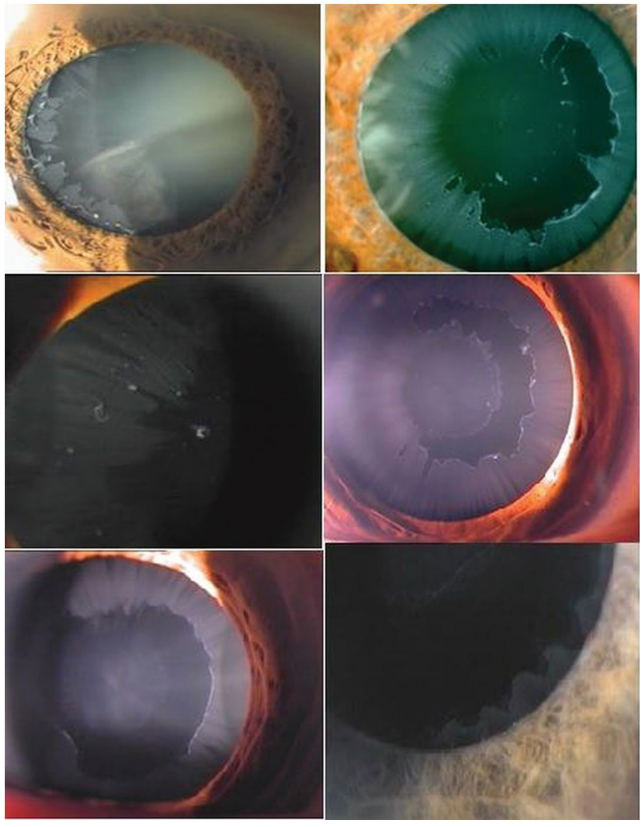

**Figure 5.** Pseudoexfoliation deposition on the anterior lens surface has a variable presentation. The classic appearance consists of a central disk, peripheral zone, and clear intermediate area separating the two areas

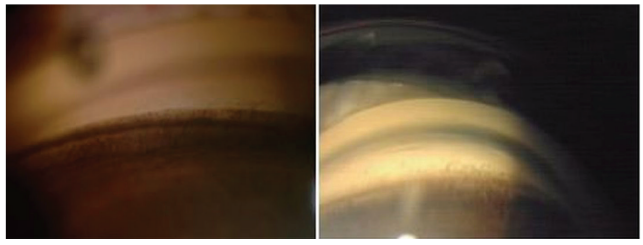

**Figure 6.** Increased trabecular pigmentation may be seen in the anterior chamber angle during gonioscopy, usually on Schwalbe's line

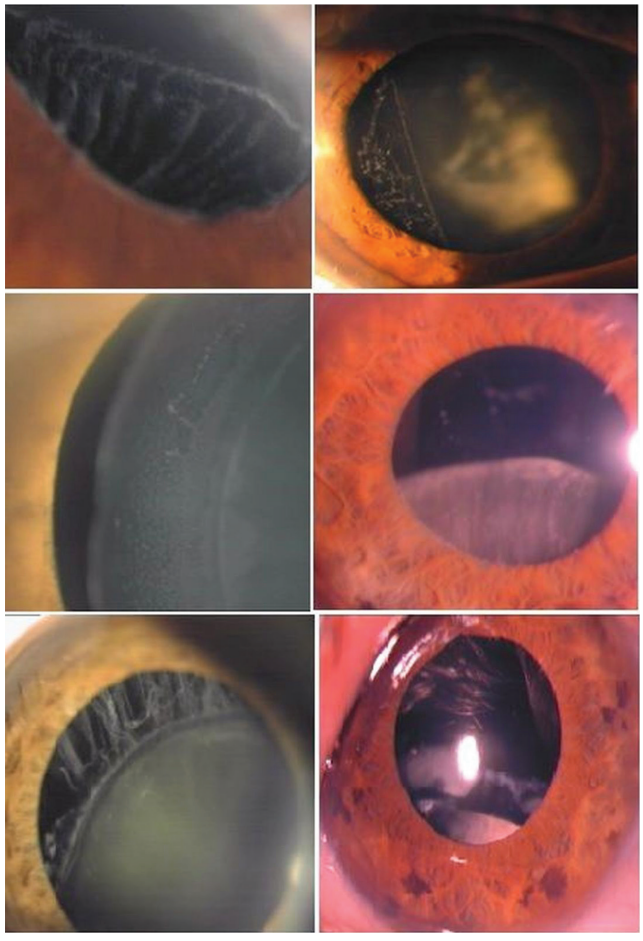

**Figure 7.** Patients with pseudoexfoliation can develop zonular weakness and lens subluxation or luxation caused by the progressive accumulation of pseudoexfoliation material

It is important that a careful preoperative clinical examination be performed after pupillary dilation. Cataract surgeons have to use more complicated instruments due to zonular weakness, inadequate pupil dilation, and blood-aqueous barrier breakdown.[48,50]

Phacoemulsification provides several advantages. Advanced surgical techniques can decrease the earlier complication rates associated with cataract surgery in PES.[51,52] Pupil dilation techniques and devices can be used to expand the pupil. A capsular tension ring may be useful in cases of significant zonular weakness.[47] A large-optic intraocular lens is recommended to compensate for the possibility of intraocular lens decentration. Postoperative complications are more common and include early postoperative IOP elevation, prolonged postoperative inflammation, posterior synechiae, and macular edema.[50] Foveal thickness in patients with PEG may be increased after uneventful phacoemulsification.[52]

Pseudoexfoliation and zonular laxity at the time of surgery are associated with late intraocular lens dislocation and anterior capsule contraction (phimosis) (Figure 9).[55,56]

PEM may be found on the anterior vitreous face and the anterior surface of the intraocular lens years after cataract extraction. Rarely, a pattern of radial striations on the anterior surface of the intraocular lens may resemble the classical pattern on the crystalline lens (Figure 10).

### Retina

PES without glaucoma may be associated with a thinner retina nerve fiber layer thickness compared to that of age-matched control subjects and non-involved fellow eyes.[57,58] Central retinal vein occlusion may be more prevalent in patients with PEG.[59]

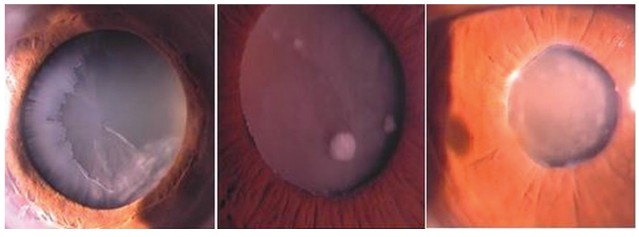

**Figure 8.** Cataract development is associated with pseudoexfoliation syndrome

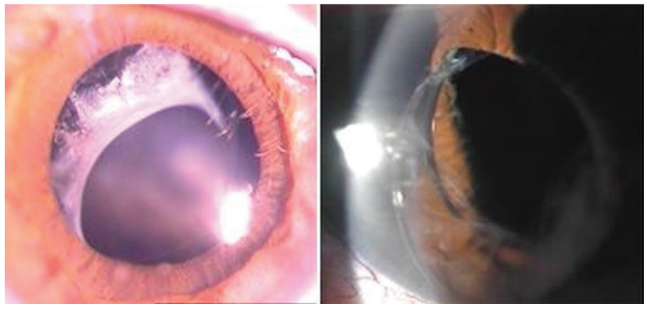

**Figure 9.** Capsule contraction syndrome and late anterior intraocular lens dislocation in a patient with pseudoexfoliation

Recently, studies using optical coherence tomography angiography demonstrated that decreased peripapillary and macular vascular density was found in pseudoexfoliation patients relative to the control group, suggesting that a vascular component, including optic nerve hypoperfusion, may be involved in the etiopathogenesis of pseudoexfoliation.[60,61,62]

The prevalence of epiretinal membrane was reported to be significantly higher in patients with PEG (19.0%) than in age-matched healthy controls (2.4%) and patients with primary open-angle glaucoma (4.1%).[63] Furthermore, more eyes with PEG had epiretinal membrane deterioration than eyes without glaucomatous alteration.[64] Both incomplete and complete posterior vitreous detachment were also more frequent in eyes with pseudoexfoliation than in fellow eyes or control eyes.[65]

### Associated Systemic Findings

PES appears to be a systemic process. Pseudoexfoliation fibers were found in autopsy tissue specimens of skin, heart, lungs, liver, kidney, and cerebral meninges in addition to the intraocular tissues.[23,66]

Cardiovascular and cerebrovascular associations reported in PES include a history of angina pectoris, systemic hypertension, stroke, asymptomatic myocardial dysfunction, impaired systemic endothelial functions, transient ischemic attacks, Alzheimer's disease, and neurosensorial hearing loss.[2,4,67,68,69,70,71,72,73,74,75,76]

PES may be a manifestation of systemic vascular disease. Middle cerebral artery blood flow velocities appear to be diminished.[77] A higher prevalence of silent ischemic brain lesions and white matter abnormalities were found by magnetic resonance imaging and diffusion tensor imaging.[78,79]

Several studies have found a relation between pseudoexfoliation and hyperhomocysteinemia which may help to explain the elevated risk of vascular diseases in PES patients.[80,81,82,83]

Increased serum antiphospholipid antibodies, a risk factor for cardiovascular and cerebrovascular disease, are more frequently seen in patients with pseudoexfoliation and glaucoma than in healthy controls and patients with POAG.[3] However, other studies found no correlation with cardiovascular and cerebrovascular diseases.[84,85]

### Pseudoexfoliation Glaucoma

PES is the most important risk factor for the development of secondary open-angle glaucoma. Approximately 30% to

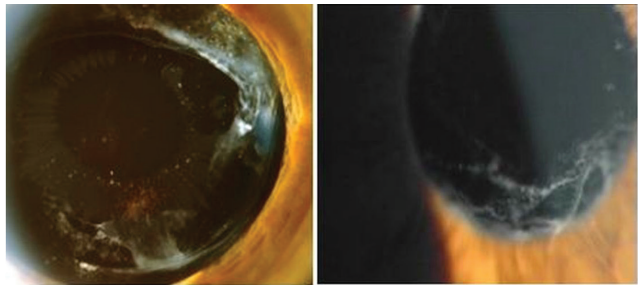

**Figure 10.** Following cataract surgery, pseudoexfoliation material deposition can be seen on the anterior vitreous face and intraocular lens

50% of patients with pseudoexfoliation develop glaucoma.[13] IOP levels and degree of pupil dilation can be important factors for developing glaucoma. At the time of diagnosis, 10-25% of patients with pseudoexfoliation have either glaucoma or elevated IOP.[13,86] Patients with PES should be followed up regularly and at short intervals with glaucoma screening tests or therapeutic interventions when appropriate.

**Differences between pseudoexfoliation and primary open-angle glaucoma**

In the past, the generally accepted clinical signs of PEG were identical to those of POAG. Now, it is important to distinguish PEG from POAG. All patients with glaucoma should be carefully examined for the clinical signs of pseudoexfoliation after pupillary dilation. PEG is clinically differentiated from POAG by the following features:

1. IOP at diagnosis is higher in PEG than in POAG.
2. IOP in PEG may rise above 50 mmHg without acute angle-closure glaucoma.
3. IOP fluctuations are wider in PEG than in POAG. A single IOP measurement is not sufficient for assessing IOP levels.
4. PEG patients may have unilateral or bilateral involvement, but asymmetric involvement is a typical feature.
5. PEG presents later than POAG.
6. PEG has more severe mean visual field defects and optic nerve head cupping than POAG.
7. PEG has more serious progression than POAG.
8. PEG is associated with a higher risk of blindness.
9. Glaucomatous damage in pseudoexfoliation patients is more related to IOP than in POAG patients.
10. PEG is more difficult to treat than POAG.
11. The IOP reduction with medical therapy is higher in PEG than in POAG.
12. Cataract formation and complications are more serious in patients with pseudoexfoliation.[13,50,87]

**Pathogenesis of pseudoexfoliation glaucoma**

In recent decades, many clinical findings have contributed to our understanding of the pathomechanisms underlying PEG. Increased outflow resistance is related to the progressive accumulation of PEM in the trabecular meshwork and Schlemm's canal cells. Subsequent degenerative changes in Schlemm's canal and juxtacanalicular tissues are causes of elevated IOP.[24]

The additional pathogenetic factor contributing to pressure elevation is melanin dispersion.[14,24]

IOP-independent factors may contribute to glaucomatous damage as well. Reported IOP-independent factors include impaired ocular and retrobulbar blood flow velocities and increased accumulation of elastic fibers in the lamina cribrosa.[88,89,90]

**Types of glaucoma**

IOP may rise over 50 mmHg despite a wide open angle. Angle-closure glaucoma may be associated with PES (Figure 11). It is a relatively rare entity. Chronic or acute angle-closure glaucoma may occur. Pseudoexfoliation is known to cause zonular weakness, anterior lens subluxation or dislocation, posterior synechia and increased iris rigidity, and occludable angles.[13,91]

In addition, neovascular glaucoma may develop after central retinal vein occlusion with PEG (Figure 12).

**Prognosis**

The time of conversion from PES to glaucoma may take years. The risk of developing glaucoma is cumulative and IOP is an important risk factor. PEG has a more severe clinical course and worse prognosis than POAG. Patients should be monitored at regular intervals.

**Treatment of PEG**

Patients with PES and no evidence of glaucoma are generally not treated, but they should be followed every six months. IOP measurements should be taken at different times of the day to determine diurnal fluctuation.

The general principles of treatment are not different from POAG but medical treatment failure in PEG is more common than in POAG patients. A general approach would be the use of medical treatment first, laser therapy second, and surgical treatment third.

**Medical therapy**

Patients with PEG often have a poorer response to medical therapy than patients with POAG. Adequate treatment requires a target IOP of 17 mmHg or lower to prevent or slow progressive damage.[93]

Prostaglandin analogs are increasingly used as the first choice of monotherapy because they are applied once daily, reduce IOP through improved outflow, and have a very low rate of systemic side effects. Travoprost and bimatoprost may provide better IOP reduction than latanoprost.[94,95]

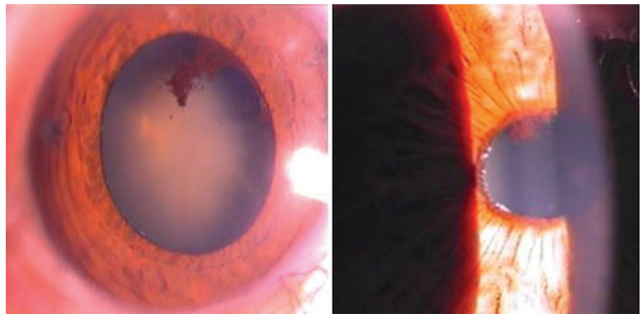

**Figure 11.** Angle-closure glaucoma in patients with pseudoexfoliation

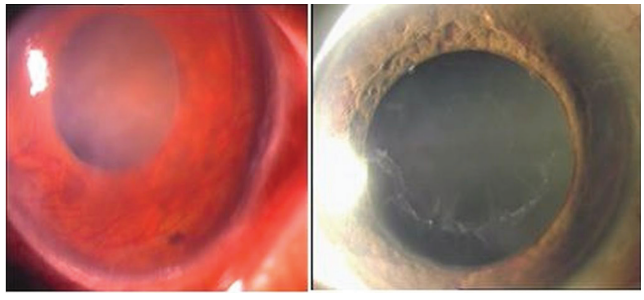

**Figure 12.** Neovascular glaucoma and pseudoexfoliation

It is often difficult to achieve target IOP with monotherapy. To prevent further glaucomatous progression, a fixed combination may be necessary as initial therapy. In a comparison of travoprost, latanoprost, and dorzolamide/timolol fixed combination, IOP lowering ranged from 8 to 11 mmHg.[96] Diurnal IOPs may be comparable with dorzolamide/timolol fixed combination and brimonidine/timolol fixed combination as first-line therapy.[97] Travoprost/timolol fixed combination may provide greater reduction compared with latanoprost/timolol fixed combination.[98] Bimatoprost/timolol fixed combination may provide greater reduction (10.2 mmHg) compared with bimatoprost alone (8.1 mmHg).[99] Poor medication compliance is common in a large proportion of PEG patients under treatment and can lead to severe deterioration of vision. Failure of optimal medical therapy occurs earlier than in open-angle glaucoma patients.

### Laser therapy

Selective laser trabeculoplasty can be effective.[100] However, lower energy settings are required due to the increased pigmentation. Additionally, the effectiveness of treatment was shown to diminish over time, and after 18 months it was still effective in only 64% of patients.[101]

### Surgery

If medication and laser therapy fail to control the progression of glaucoma, surgery may be performed with comparable success rates to POAG. Because patients with PEG have higher IOP at diagnosis, they tend to undergo glaucoma filtering surgery more frequently than patients with POAG.[14]

Trabeculectomy with mitomycin-C has better IOP control than successful maximal medical therapy in advanced disease.[94,102] Postoperative inflammatory responses, fibrinous reactions, and posterior synechia formation are higher in PEG patients.

Combined cataract extraction and trabeculectomy are performed more frequently for managing patients with visually significant cataract and PEG than POAG. Furthermore, it has been shown that phacotrabeculectomy has similar success rates as trabeculectomy and can be as safe and effective as trabeculectomy in the long-term follow-up of both PEG and POAG patients.[103] Non-penetrating glaucoma surgery may avoid some of the complications associated with trabeculectomy. IOP reduction may be less than with perforating surgery.[104,105,106]

Recently, minimally invasive glaucoma surgery offers results in patients with mild to moderate glaucoma with the advantage of a safe risk profile.[107,108] Studies have also shown that gonioscopy-assisted transluminal trabeculotomy provides effective IOP reduction in PEG.[109,110]

## Conclusions

PES and glaucoma are public health problems in older age worldwide. In recent years, the expansion in knowledge of the epidemiology, pathogenesis, and genetics of PES and glaucoma has provided important insights for understanding this disease and future treatment.

Pseudoexfoliation is an age-dependent and stress-induced extracellular fibrotic matrix disorder in which the oxidative-antioxidative balance is disturbed. *LOXL1* is involved in the pathogenesis. Patients with PES have a higher risk of cardiovascular and cerebrovascular diseases.

The presence of pseudoexfoliation is an important risk factor for glaucoma and cataract. It is necessary to distinguish PEG from POAG. Medical management is more difficult and surgery is required more frequently. Early diagnosis, appropriate treatment and more frequent examinations appear to prevent the progression of visual field and vision loss.

In the future, new research can increase our understanding of the epidemiology, pathogenesis, genetics, and classifications of this disease. New therapeutic approaches will be considered for its management.

### Ethics

### Authorship Contributions

Concept: N.Y., Design: N.Y., Data Collection or Processing: N.Y., B.Y.T., Analysis or Interpretation: N.Y., B.Y.T., Literature Search: N.Y., B.Y.T., Writing: N.Y., B.Y.T.

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