InSight Bulletin: A Multidisciplinary Interlink International Research Journal
Peer Reviewed International, Open Access Journal.
ISSN : 3065-7857 / Website: https://ibrj.us / Volume-2, Issue-4 / April - 2025

Original Article

Pigment Dispersion Syndrome: An Overview

Sakshi Swapnil Bhanap
Deparment of Pharmaceutics,D.S.T.S.Mandal's college of pharmacy ,Solapur

Manuscript ID:
IBMIIRJ -2025-020404

Submitted: 02 Mar 2025

Revised: 16 Mar 2025

Accepted: 03 Apr 2025

Published: 30 Apr 2025

ISSN: 3065-7857

Volume-2

Issue-4

Pp. 18-24

April 2025

Correspondence Address:
Sakshi. Swapnil. Bhanap
Deparment of Pharmaceutics,D.S.T.S.Mandal's college of pharmacy ,Solapur
Email: *sakshi.bhanap@gmail.com*

OPEN ACCESS

Quick Response Code:

Web. https://ibrj.us

DOI: 10.5281/zenodo.15964783

DOI Link:
https://doi.org/10.5281/zenodo.15964783

Creative Commons

Abstract :

Pigment dispersion syndrome (PDS) is an ocular condition defined by the release and subsequent dispersion of pigment granules from the iris pigment epithelium throughout the anterior segment of the eye. This process arises from the mechanical rubbing of the posterior iris surface against the lens zonules, leading to pigment shedding. The released pigment circulates within the aqueous humor and accumulates on various ocular structures, most notably the trabecular meshwork, the eye's drainage system. Clinically, PDS presents with characteristic signs, including iris transillumination defects, a Krukenberg spindle (pigment deposition on the corneal endothelium), and increased pigmentation of the trabecular meshwork. Notably, PDS is often associated with myopia and is more prevalent in young, Caucasian males. A significant concern is the potential progression to pigmentary glaucoma (PG), a secondary open-angle glaucoma, where pigment accumulation obstructs aqueous humor outflow, elevating intraocular pressure (IOP) and potentially damaging the optic nerve. Diagnosis relies on thorough slit-lamp examination and gonioscopy to visualize these signs. Management primarily focuses on lowering IOP through medications, laser trabeculoplasty, or surgery. As PDS can be asymptomatic, regular eye examinations are crucial for early detection and intervention to prevent PG and preserve visual function. Ongoing research also explores the genetic components that may contribute to PDS.

Keywords: *Pigment dispersion syndrome, Pigmentary glaucoma, Iris posterior bowing, Intraocular pressure*

Introduction:

Melanin pigment granules that circulate in the aqueous humor (AH) and gather on various systems within the anterior chamber (AC) of the eye, which include the corneal endothelium, lens floor, zonules, iris, iridocorneal perspective, and trabecular meshwork (TM), are the hallmark of pigment dispersion syndrome (PDS)[1]. reduced aqueous outflow can result from pigment granule accumulation inside the angular systems and TM, which raises intraocular strain (IOP). PDS may additionally bring about ocular high blood pressure (OHT) and a secondary boom in intraocular strain (IOP), which may additionally damage the optic nerve and result in the development of pigmentary glaucoma (PG), a secondary shape of glaucoma. whilst glaucomatous optic neuropathy, retinal nerve fiber layer (RNFL) thinning, and/or sight view abnormalities are introduced on with the aid of excessive IOP, peripheral vision (PG) might also result [2]. In actuality, PDS and PG constitute various tiers of severity alongside a continuum of diseases. PDS is a massive risk issue for the emergence of PG and OHT three. even though glaucoma can take many exceptional paperwork, number one open-attitude glaucoma (POAG) is the most commonplace kind. Open-angle glaucoma is idea to have a secondary form in PG [1-2]. PDS is commonly diagnosed clinically with the aid of various symptoms and signs, along with pigmentation in TM, radial mid-peripheral transillumination iris abnormalities, and small pigment deposits on the principal portion of the corneal endothelium [2-3]. The mechanisms underlying PDS formation are because of the non-stop rubbing and friction that takes place throughout scholar actions between the pigment iris epithelium and lens additives. these moves normally occur while the iris is posteriorly bent and opposite pupillary blocked three.

studies have proposed that some sufferers have a burn-out section or pigment reversal later in lifestyles, all through which time PDS or PG symptoms may be much less substantial.In phrases of historic standards, the presentation of vertical pigment accumulation at the corneal endothelium turned into in the beginning described via Friedrich Krukenberg in 1899 [4]. This situation is now called a Krukenberg spindle.

Creative Commons (CC BY-NC-SA 4.0)
This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International Public License, which allows others to remix, tweak, and build upon the work noncommercially, as long as appropriate credit is given and the new creations ae licensed under the idential terms.

How to cite this article:
Bhanap, S. S. (2025). Pigment Dispersion Syndrome: An Overview. Insight Bulletin: A Multidisciplinary Interlink International Research Journal, 2(4), 18–24. https://doi.org/10.5281/zenodo.15964783

Von Hippel later linked this scientific characteristic to glaucoma in 1901, claiming that pigment buildup brought about AH outflow blockage [5]. Sugar said the first example of PG in 1940[6]. nine years later, the identical writer stated OHT, pigment buildup in the TM, transillumination iris abnormalities, and Krukenberg spindle in 2 male myopes with PG[7]. After locating pigment within the TM, Levinsohn postulated that the iris changed into the supply [8,9].

There are extra than seventy five million glaucoma patients globally, divided between PDS and PG patients. In individuals among the a long time of 40and 80, the prevalence is three.5%; by way of 2040, it's far anticipated to upward push to nearly one hundred ten million [10]. In 1–1.5 percentage of cases in Caucasian international locations, glaucoma is due to PG and PDS [1]. PDS and PG are extra common in Caucasian patients with myopia between 20 and forty years of age with an even distribution among men and women for PDS and a minor a touch male predominance for PG[11,12]. With an entire life conversion charge of 10–50%, PDS is a risk issue for developing PG, in particular whilst mixed with OHT and anatomic myopic predisposition[1,13 -14]. This succinct summary objectives to provide medical doctors a fast refresher on key topics and key takeaways to preserve in thoughts whilst diagnosing, treating, and coping with sufferers with PDS. progressed knowledge of PDS is critical for the differential diagnosis, management, and remedy of people with PG and PDS which will save you irreparable structural and functional damage from glaucomatous sickness from growing and/or progressing [15].

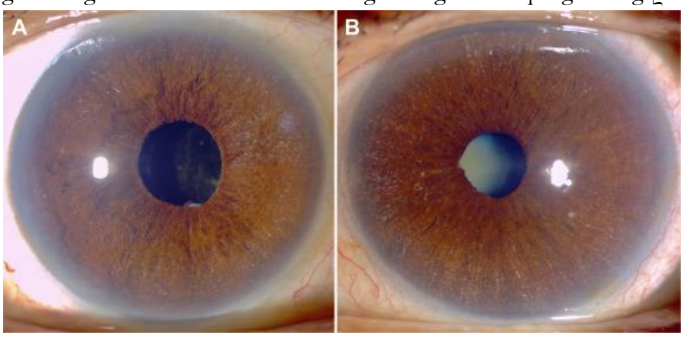

Figure.1. Difference between normal eye (A) and dispersion pigment syndrome eye (B)

Pathophysiology And Clinical Feature :

Pigment release and accumulation in AC structures are the root causes of PDS, and they can also improve IOP and result in PG. Atrophy or degeneration of the iris pigment epithelium (IPE) [16-17] or pigment loss from the iris due to congenital mesodermal dysgenesis 15 were the primary theories approximately the congenital etiology of PDS and PG. The family existence of Krukenberg spindle has been defined by way of capacity genetic causes [18]. no matter the low incidence of familial PG and PDS, studies has revealed a ability multifactorial pattern of inheritance and autosomal dominant inheritance for PDS , which may also have an effect on how iris coloration, gender, and refractive error appear clinically. [19.20]A capability PDS gene on chromosome 7Q35-q36 changed into diagnosed by way of Anderson et al. [21] based totally on an autosomal dominant sample seen in sufferers from four PDS-affected Irish families. numerous genetic loci, inclusive of Glycoprotein nmb (GpnmbR150x), Gene GPDS1 (glaucoma-associated PDS 1), and (OMIM identification 600510) had been related to PDS in studies[22]. the principle motive of the pigment showers inside the AC is friction and rubbing between the IPE and the posterior floor and zonules of the lens.

This friction and rubbing is made worse by using reverse pupillary block mechanisms brought on by using multiplied iridolenticular contact and with the aid of the backward posterior bowing of the iris, that's present in reasonably myopic eyes with extra space [23,24]. in line with ultrasonographical research , physiological processes such accommodation, blinking, eye actions, head orientations, and exercising would possibly motive elevated friction and speak to between AC systems, which favors opposite pupillary block[25]. In sure occasions, the AH actions from the posterior chamber (computer) to the AC in a unidirectional mode, developing a high pressure inside the AC that favors similarly apposition among the iris and lens surface. This ball-valve mechanism may be as a result of the increased contact among the iris and lens structures in eyes which are prone to having a deep AC and/or a massive iris [26] .The AH trapped inside the AC can create posterior bending and extended friction among the peripheral posterior iris and zonules and lens systems leading to pigment dispersion showers. regarding the common characteristics and effects of treatment for PG, a large meta-analysis based on 5 randomized managed trials become done in a Cochrane review in 2016 [27]. The dispersion of melanin granules (at the lens, iris, cornea, and TM) and friction among anatomic additives in the AC are a few of the common aspects of PDS that had been stated inside the have a look at. Pigment granules which have been deposited may additionally motive damage to the TM, which may also result in an accumulation of AH and a high IOP at the side of glaucomatous optic neuropathy. Topical medicinal drug has a tendency to be the primary-line treatment, corresponding to different varieties of glaucoma. Peripheral iridotomy was now not supported via enough proof to be used as a treatment for PG, in line with the Cochrane evaluation. more research is needed to assess the scientific software of this laser treatment in PDS and PG sufferers.

PDS is usually asymptomatic and located for other reasons throughout a normal or pressing ocular checkup. some sufferers have pronounced experiencing headaches, halos surrounding vivid sources, and blurring episodes, specially after physical interest, prolonged reading, or unique head motions brought on with the aid of OHT spikes that PDS prefers [28-29]. Pigment showers in the AC, Krukenberg spindle, iris trans-illumination abnormalities, and accelerated pigmentation in the TM are the various medical manifestations of PDS . Myopia and a noticeably deep AC are not unusual in PDS sufferers, which beautify the iris's backward bowing in the direction of the zonules and reverse pupillary block mechanisms [24, 25]. OHT, which could grow to be PG within the presence of RNFL thinning, optic neuropathy, and/or glaucomatous field of vision abnormalities, may be due

to PDS. Pigment showers in the AC end result from melanin pigments produced due to the continual mechanical contact among the IPE and zonule systems in prone eyes. PDS may also or may not have vertically orientated deposits at the corneal endothelium known as Krukenberg spindles [1]. according to histological investigations, melanin granules are visible inside corneal endothelial cells, indicating that pigment phagocytosis takes place rather than only pigment being deposited on the cornea [30-31]. it has been confirmed that endothelial mobile numbers and characteristic in PDS patients are comparatively normal, with ability pleomorphism and polymegathism [32].

A less common characteristic this is on occasion left out in PDS and PG eyes has also been stated in some of case reports [33-34]. at the posterior lens pill, important deep pigment deposits are also visible near the equator [35-36]. those deposits are maximum possibly the end result of anatomic abnormalities in the Wiegers ligament and a conversation between the posterior chamber and the posterior lens pill [37–38].studies have verified that the AC of sufferers with PDS tended to be deeper whilst compared to affected person age-and refraction-corrected individuals with POAG[39]. these sufferers can also have an iris that actions backwards and touches the lens structure because of a larger AC space. whilst mild is directed via the student perpendicularly, iris trans-illumination defects with a spoke-like sample are visible because of the pigment launched by the IPE. these are more substantive in eyes with mild-coloured irises and are found in over 85% of human beings with PDS thirteen. The iris frequently famous pigment that might accumulate in the iris furrows on the anterior location[19]. Anisocoria, hyperplastic dilator muscle groups, heterochromia, mydriasis, and pigment loss of the iris can end result from the IPE's non-stop rubbing and friction with the zonule systems [1]. Campbell in the beginning noted the potential etiology of PDS patients' posterior iris bowing in 1979 [40]. those eyes generally have a wide iris which could facilitate irido-lenticular rubbing and friction via bending backward inside the mid-peripheral vicinity while a deep AC is gift. opposite pupillary block can result in a ball-valve mechanism in which AH moves from computer to AC unidirectionally, creating OHT and elevated AC pressure that promote iris motion backward and enhance contact among IPE and zonule lenticular systems [24, 26]. exercise, lodging, blinking, and head movements are examples of physiological interest that might activate episodes of a reverse pupillary block , which might also account for the episodic headache and blur signs and symptoms that some PDS patients revel in those with PDS commonly showcase multiplied TM pigmentation on gonioscopy 20. In comparison to the patchy-patterned dark pigmentation seen in people with pseudoexfoliation syndrome (PEX), which might aid within the differential prognosis, the dark pigmentation in TM patients has a tendency to be uniform, full circumference, and major inferiorly, maximum possibly due to gravity [41,42]. Histological investigations have confirmed the lifestyles of phagocytosed pigment granules in TM endothelial cells, corresponding to the corneal endothelium[43]. a reduced outflow of AH, OHT, and PG may additionally end result from a extended excess of phagocytosed pigment in the TN, that's taken into consideration to motive apoptosis, mobile necrosis, and anatomic structural modifications of this shape [44,45]. it's miles essential to remember the fact that the PDS diagnosis is clinical and can be tough due to the massive variety of medical characteristics. the following signs ought to suggest PDS: pigment deposition at the endothelium (Krugenberg spindle); pigment granules on the iris; concave peripheral iris configuration; spoke-like transillumination defects of the iris; pigment deposition at the anterior lens capsule; pigment deposition on the posterior lens tablet (Zentmeyr line, additionally seen in more current observations at the more valuable part of the posterior lens capsule); and pigmentation of TM the use of gonioscopy (line similar to Sampaolesi's line).

Diffrential Diagnosis :

PEX can result in TM pigmentation that looks patchy, with deposits within the AC that resemble dandruff-like flakes as opposed to darkish pigment fabric. Trans-illumination defects of the iris can also end result from PEX, but these tend to be positioned alongside the pupillary margin rather than the mid-periphery of the iris [36]. due to the reduced outflow of AH in TM, PEX and PDS are full-size threat factors for OHT. Pseudoclus glaucoma and PG can end result from sight view loss glaucomatous optic neuropathy, and RNFL weakening. Iris defects, excessive IOP, deposits within the AH, and uveitis can all result from trauma, ocular surgical operation, contamination, and uveitis forty five. Pigment showers may be caused by inflammatory cells and debris from positive ocular illnesses. After cataract surgical operation, the differential diagnosis have to don't forget Uveitis-Glaucoma-Hyphema syndrome, additionally referred to as Ellingson syndrome. This uncommon hassle following intraocular lens implants can result in iris transillumination abnormalities, pigmentary dispersion, excessive IOP, and hyphema [46]. sure intraocular malignancies, such uveal melanomas, can purpose pigment deposits, iris trans-illumination abnormalities, and OHT, however they're rarely discovered [47]. clinical manifestations of non-transparent AH, deposits, iris abnormalities, and IOP spikes can also be seen in different issues inclusive of Horner syndrome, Posner Schlossman syndrome, cataract surgical procedure, rhegmatogenous retinal detachment, diabetes, and lengthy-term mydriasis drug use. an intensive ophthalmological exam, and correct and whole history, coupled with instrumental tests if suitable, can help within the differential diagnosis to identify the proper custom designed control and treatment. exams including ultrasonic biomicroscopy, optical coherence tomography, and field of vision checking out can resource in prognosis and treatment.

Management And Treatment :

PDS is a threat element for PG, wherein OHT may emerge because of the faded and less efficient outflow mechanisms of the AH with the aid of the dispersion of pigment that causes irreversible functional and structural harm. the only glaucoma treatment aimed toward decreasing IOP is laser therapy, local drop therapy, or surgical operation. remedy won't be essential if PDS does no longer show off expanded IOP ranges, but it's far nevertheless critical to have normal ophthalmologic examinations and, if essential, instrumental trying out. because of the massive amount of pigment inside the iris's intermediate pigment epithelium (IPE layer), pigment dispersion poses a severe hazard to visible function in humans of coloration, Asians, and Africans. inside the pigmented races, greater energetic and vigorous interference is necessary due to reverse pupillary obstruction and

pigment dispersion. The duration of time between follow-up appointments ought to be decided case-with the aid of-case, deliberating factors along with age, coexisting ophthalmologic issues, other chance elements, ailment severity, and so forth.

a number of studies have cautioned staging the illness to ascertain the correct stage of competitive therapy and control 2. Richter and associates delineated four stages, thinking of the IOP and pigment dispersion interest in PDS. those levels incorporate: (1) solid IOP whilst there's inactive pigment dispersion; (2) strong IOP when there may be energetic pigment dispersion; (three) PG and OHT whilst there is energetic pigment dispersion; and (4) PG and OHT or ordinary IOP when there's inactive pigment dispersion. To assist manipulate PDS, 3 medical ranges were proposed lately three[48,49]. those are: (1) asymptomatic pigment dispersion resulting from physiological elements like workout, lodging, and stress that don't result in OHT; (2) PG with noticeable pigment in TM and excessive IOP; and (3) glaucomatous functional and morphologic harm, which incorporates pigment clearing from TM with normalizing IOP stages that normally arise with growing older. in step with Gandolfi et al. [50], the range of pigmented particles released at some stage in provocative checking out with phenylephrine in sufferers with PDS can help become aware of those sufferers who are at a better risk of developing extended IOP over time. these sufferers may additionally gain from preventative laser peripheral iridotomy (LPI), which broadly speaking entails inducing iris knocking down to lessen concavity, posterior iris bowing, and opposite pupillary block. it's far crucial to keep in mind that even if sure staging strategies have been suggested by means of studies, doctors may not usually help and employ them whilst treating sufferers in an average medical context.

usually, PDS does no longer translate to PG and does not specific itself until adulthood. PDS is a hazard thing; nevertheless, multiple longitudinal studies suggest that maximum PDS eyes in no way expand PG [14]. The ability of the TM to accept or take away pigment particles and alter AH outflow methods can be partially answerable for the discrepancy. but, extraordinary research have determined varying conversion costs, which in certain instances can reach 50%. 10% of PDS eyes advanced PG after 5 years, and 15% advanced PG after fifteen years, in line with Siddiqui et al. [13].

whilst treating PDS, an individual's age is a crucial consideration. sure experts suggest that positive patients may undergo a burn-out segment later in lifestyles wherein warning signs of PDS or PG are much less seen. Pigment reversal signal is another call for it. In those patients, the inferior TM commonly clears first, and the IOP can normalize and less pigment is visible in the TM 1. every so often, on this stage of burnout, PG patients with ordinary IOP stages display a discount in IOP-reducing medication. according to a few writers, PG sufferers can be incorrectly diagnosed with POAG or regular-tension glaucoma due to the presence of much less pigment in the TM and a regular IOP three. concerning the effect of race, it's far important to understand that homogenous TM pigmentation, spoke-like iris transillumination abnormalities, and the Krukenberg spindle are commonplace and classic observations in Caucasians. then again, iris transillumination deficiencies are normally nonexistent inside the pigmented races. however, PDS poses a serious risk to imaginative and prescient in pigmented races including Asians and people of coloration because of the huge extent of pigment granules in the IPE layer. this could lead to intense TM pigmentation and fast blockage of the AH outflow, which can cause consistently high intraocular pressure, glaucomatous neuropathy, and blindness. In pigmented PDS sufferers, the burnout section is hardly ever located in medical exercise.

PDS is normally identified as a result of a standard ocular exam. most of the time, the sufferers are asymptomatic and are available to the health practitioner for a prescription for glasses or an eye exam. A slit-lamp exam exhibits the spoke-like iris transillumination abnormalities together with the scientific signs of pigment deposits in the AC structures and cornea. recently, a novel and simple method for identifying spoke-like patterns inside the iris the use of an automated refractometer has been described [51]. This method is straightforward to apply and can be useful in ordinary scientific settings. With gonioscopy, pigment buildup may be observed. The identical tools and techniques used to deal with glaucoma also are used to diagnose and treat PDS conversion to PG, which includes optical coherence tomography, field of vision evaluation, and tonometry. Decreasing IOP to intention values in ranges that prevent or sluggish irreversible glaucomatous morphology and visual field harm is the cornerstone of glaucoma treatment and care. surgical procedure, lasers, and IOP-reducing drops are feasible forms of therapy. One giant hazard thing for the development of PG is PDS. IOP elevation in PDS/PG is dealt with within the identical manner as other varieties of glaucoma. local remedy for expanded IOP and glaucoma includes topical carbonic anhydrase inhibitors, prostaglandins, beta-blockers, alpha-agonists, and miotics. Miotics are normally not well tolerated. In individuals with PDS/PG, beta-blockers and prostaglandins have demonstrated efficacious effects in decreasing IOP 2. The iris and/or TM may be treated with laser treatment. to improve outflow on the TM, argon and selective laser trabeculoplasty, or SLT and ALT, respectively, may be implemented. younger patients with PG have reportedly found ALT to be helpful , but over the years, success quotes generally tend to decline, most in all likelihood due to pigment launch, overtreatment, immoderate TM electricity absorption, and irreversible tissue scarring. SLT is an fascinating opportunity for those patients in idea, specifically as it may be repeated over the years and reveals a bigger, more efficient, and selective energy absorption in tissues with pigment. however, studies on SLT in PG discovered that fulfillment prices of 85% at 365 days decreased to fourteen% after 4 years, with a failure fee at roughly 27 months [52,53]. moreover, SLT must be used cautiously in PG sufferers who've formerly acquired treatments with ALT, have deeply pigmented TM, or are using numerous glaucoma IOP-reducing capsules as it has been tested to cause IOP rises following remedy, especially in those with closely pigmented TM [54].

The morphological and anatomical predisposition of PDS eyes vulnerable to OHT has been addressed in some of studies through LPI 20, 40. that is mostly carried out by inducing iris flattening to lessen concavity, posterior iris bowing, and opposite pupillary block, which together create a right equilibrium of AH float from the computer to AC. however, whilst compared to eyes that did now not get hold of treatment, numerous long-time period systematic studies and critiques 27,have established that LPI had minimum advantage and few medical variations in danger of IOP rises[55,56-57]. LPI can be considered in very unique cases in young people who show irido-zonular contact with UBM with regular IOP [58-59]. while target pressures aren't reached with laser remedy or maximal IOP-decreasing drop therapy, or in patients who can not tolerate drops, surgical procedure can be considered[60-61]. surgical procedure is generally considered as a backup or 1/3 alternative due to its inherent risks and

invasiveness. traditional trabeculectomy, like other styles of glaucoma, is useful in reducing IOP, but surgical risks are still full-size. even though they have got decrease surgical risks, alternative surgical approaches encompass canaloplasty, goniotomy, Ab interno trabeculectomy, trabectome, minimally invasive glaucoma surgical treatment strategies, and current implants have lower fulfillment rates in achieving IOP target levels [62]. while surgical treatment is needed, every patient's state of affairs must be evaluated to determine the satisfactory path of movement.

Conclusion :

PDS is a as a substitute frequent situation with a diverse range of medical traits. For each people and society as an entire, early identification, affected person education, and treatment of sufferers at hazard are essential. commonly, it's far found in the course of recurring ophthalmologic appointments. maximum sufferers do now not have any signs and symptoms. Preventive eye exams are therefore quite vital. humans with OHT and PG ought to searching for treatment right away to save you irreparable morphological and useful damage from glaucoma and its modern progression through the years. stop-level glaucoma and different sight-threatening issues may be prevented and the want for severe healing procedures decreased with the assist of patient schooling, correct diagnosis, and effective scientific care.

Abbreviation :

1. AH: Aqueous chamber
2.AC:Anterior chamber
3.TM; Trabecular meshwork
4.PDS;Pigment dispersion syndrome
5IOP:Intraocular pressure
6.OHT:Ocular hypertension
7.PG:Pigmentary glaucoma
8.RNFL:Retinal nerve fibre layer
9`POAG:Primary open angle glaucoma

Acknowledgements:

I would like to express my special thanks of gratitude to the management of D.S.T.S.Mandal's College of Pharmacy ,Solapur,India I would also like to thank teaching staff and parents and friends who helped me a lot in finalizing this review paper on

Pigment Dispersion Syndrome : An Overview.

Conflict Of Interest:

The authors declare no conflicts of interest.

References :

1. Niyadurupola N, Broadway DC. Pigment dispersion syndrome and pigmentary glaucoma--a major review. *Clin Exp Ophthalmol.* 2008;36:868–82. [PubMed] [Google Scholar]
2. Okafor K, Vinod K, Gedde SJ. Update on pigment dispersion syndrome and pigmentary glaucoma. *Curr Opin Ophthalmol.* 2017;28:154–60. [PubMed] [Google Scholar]
3. Scuderi G, Contestabile MT, Scuderi L, Librando A, Fenicia V, Rahimi S. Pigment Dispersion Syndrome and Pigmentary Glaucoma:A Review and Update. *Int Ophthalmol.* 2019;39:1651–62. [PubMed] [Google Scholar]
4. Krukenberg F. Beiderseitigeangeborene Melanse Der Hornhaut. *Klin Monatsbl Augenbeilkd.* 1899;37:254 8. [Google Scholar]
5. Von Hippel E. Zur Pathologischen Anatomie Des Glaucoma. *Arch Ophthalmol.* 1901;52:498. [Google Scholar]
6. Sugar HS. Concerning the Chamber Angle I. Gonioscopy. *Am J Ophthalmol.* 1940;23:853–66. [Google Scholar]
7. Sugar HS, Barbour FA. Pigmentary Glaucoma:A Rare Clinical Entity. *Am J Ophthalmol.* 1949;32:90–2. [PubMed] [Google Scholar] Review. *Am J Ophthalmol.* 1966;62:499–507. [PubMed] [Google Scholar]
8. 8.Zhou R, Tang Q, Pu L, Qing G. Changes of trabecular meshwork pigmentation in patients with pigment dispersion syndrome: A 15-year study. Medicine (Baltimore). 2021 Aug 06;100(31):e26567. [PMC free article] [PubMed]
9. Levinsohon G. Beitrag Zurnpathologisch e Anatomie und Pathologie des Glaukoms. *Arch Augenbeilkd.* 1909;62:131. [Google Scholar]
10. Jonas JB, Aung T, Bourne RR, Bron AM, Ritch R, Panda-Jonas S. Glaucoma. *Lancet.* 2017;11390:2183–93. [PubMed] [Google Scholar]
11. Zeppieri M, Tripathy K. *StatPearls.* Treasure Island FL: StatPearls Publishing; 2022. Pigment Dispersion Glaucoma. [PubMed] [Google Scholar]
12. Farrar SM, Shields MB. Current Concepts in Pigmentary Glaucoma. *Surv Ophthalmol.* 1993;37:233–52. [PubMed] [Google Scholar]
13. Siddiqui Y, Ten H, ulzen RD, Cameron JD, Hodge DO, Johnson DH. What is the Risk of Developing Pigmentary Glaucoma from Pigment Dispersion Syndrome? *Am J Ophthalmol.* 2003;135:794–9. [PubMed] [Google Scholar]
14. Mastropasqua L, Ciancaglini M, Carpineto P, Gallenga PE. Early Standiation of Pigmentary Dispersion Syndrome and Long-term Analysis of Progression to Pigmentary Glaucoma. *Ann Ophthalmol Glaucoma.* 1996;28:301–7. [Google Scholar]
15. Lichter PR. Pigmentary Glaucoma:Current Concepts. *Trans Am Acad Ophthalmol Otolaryngol.* 1974;78:309–13. [PubMed] [Google Scholar]
16. Kupfer C, Kuwabara T, Kaiser-Kupfer M. The Histopathology of Pigmentary Dispersion Syndrome with Glaucoma. *Am J Ophthalmol.* 1975;80:857–62. [PubMed] [Google Scholar]

17. Fine BS, Yanoff M, Scheie HG. Pigmentary "Glaucoma":A Histologic Study. *Trans Am Acad Ophthalmol Otolaryngol.* 1974;78:314–25. [PubMed] [Google Scholar]
18. 18.Mauksch H. Über Idiopathischen Zerfall des Retinalen Pigmentblattes der Iris bei Zwei Brudern. *Z Augenheilkd.* 1925;57:262. [Google Scholar]
19. McDermott JA, Ritch R, Berger A, Wang RF. Inheritance of Pigmentary Dispersion Syndrome. *Invest Ophthalmol Vis Sci.* 1987;28:153. [Google Scholar]
20. Scheie HG, Cameron JD. Pigment Dispersion Syndrome:A Clinical Study. *Br J Ophthalmol.* 1981;65:264–9. [PMC free article] [PubMed] [Google Scholar]
21. Andersen JS, Pralea AM, DelBono EA, Haines JL, Gorin MB, Schuman JS, et al. A Gene Responsible for the Pigment Dispersion Syndrome Maps to Chromosome 7q35-q36. *Arch Ophthalmol.* 1997;115:384–8. [PubMed] [Google Scholar]
22. Lascaratos G, Shah A, Garway-Heath DF. The Genetics of Pigment Dispersion Syndrome and Pigmentary Glaucoma. *Surv Ophthalmol.* 2013;58:164–75. [PubMed] [Google Scholar]
23. 23.. Campbell DG, Schertzer RM. Pathophysiology of Pigment Dispersion Syndrome and Pigmentary Glaucoma. *Curr Opin Ophthalmol.* 1995;6:96–101. [PubMed] [Google Scholar]
24. Ritch R. A Unification Hypothesis of Pigment Dispersion Syndrome. *Trans Am Ophthalmol Soc.* 1996;94:381–409. [PMC free article] [PubMed] [Google Scholar]
25. Potash SD, Tello C, Liebmann J, Ritch R. Ultrasound Biomicroscopy in Pigment Dispersion Syndrome. *Ophthalmology.* 1994;101:332–9. [PubMed] [Google Scholar]
26. Karickhoff JR. Pigmentary Dispersion Syndrome and Pigmentary Glaucoma:A New Mechanism Concept, a New Treatment, and a New Technique. *Ophthalmic Surg.* 1992;23:269–77. [PubMed] [Google Scholar]
27. Michelessi M, Lindsley K. Peripheral Iridotomy for Pigmentary Glaucoma. *Cochrane Database Syst Rev.* 2016;2:CD005655. [PMC free article] [PubMed] [Google Scholar]
28. Schenker HI, Luntz MH, Kels B, Podos SM. Exercise-induced Increase of Intraocular Pressure in the Pigmentary Dispersion Syndrome. *Am J Ophthalmol.* 1980;89:598–600. [PubMed] [Google Scholar]
29. Haynes WL, Johnson AT, Alward WL. Inhibition of Exercise-induced Pigment Dispersion in a Patient with the Pigmentary Dispersion Syndrome. *Am J Ophthalmol.* 1990;109:601–2. [PubMed] [Google Scholar]
30. Iwamoto T, Witmer R, Landolt E. Light and Electron Microscopy in Absolute Glaucoma with Pigment Dispersion Phenomena and Contusion Angle Deformity. *Am J Ophthalmol.* 1971;72:420–34. [PubMed] [Google Scholar]
31. Korobova V. On the Etiology of Krukenberg's Spindle. *Russkii Ophth J.* 1929;476:84. [Google Scholar]
32. Lehto I, Ruusuvaara P, Setala K. Corneal Endothelium in Pigmentary Glaucoma and Pigment Dispersion Syndrome. *Acta Ophthalmol (Copenh)* 1990;68:703–9. [PubMed] [Google Scholar]
33. Canestraro J, Sherman J. Curvilinear, Symmetrical, and Profound Pigment Deposition on the Posterior Lens Capsule in a Patient with Bilateral Pigmentary Dispersion Syndrome. *Eye Brain.* 2018;10:79–84. [PMC free article] [PubMed] [Google Scholar]
34. Al-Mezaine HS. Central Posterior Capsule Pigmentation in a Patient with Pigment Dispersion and Previous Ocular Trauma:A Case Report. *Indian J Ophthalmol.* 2010;58:336–7. [PMC free article] [PubMed] [Google Scholar]
35. Nagarajaiah S, Shun-Shin GA. Pigment Deposition on the Central Aspect of the Posterior Lens Capsule in Pigmentary Dispersion. *Digit J Ophthalmol.* 2011;17:69–71. [PMC free article] [PubMed] [Google Scholar]
36. Trampuž IŠ. A Case of Dense Pigment Deposition of the Posterior Lens Capsule. *BMC Ophthalmol.* 2020;20:458. [PMC free article] [PubMed] [Google Scholar]
37. Turgut B, Türkçüoğlu P, Deniz N, Catak O. Annular and Central Heavy Pigment Deposition on the Posterior Lens Capsule in the Pigment Dispersion Syndrome:Pigment Deposition on the Posterior Lens Capsule in the Pigment Dispersion Syndrome. *Int Ophthalmol.* 2008;28:441–5. [PubMed] [Google Scholar]
38. Lin DY, Volpicelli M, Singh K. Dense Pigmentation of the Posterior Lens Capsule Associated with the Pigment Dispersion Syndrome. *J Glaucoma.* 2003;12:491–3. [PubMed] [Google Scholar]
39. Caprioli J, Spaeth GL, Wilson RP. Anterior Chamber Depth in Open Angle Glaucoma. *Br J Ophthalmol.* 1986;70:831–6. [PMC free article] [PubMed] [Google Scholar]
40. Campbell DG. Pigmentary Dispersion and Glaucoma:A New Theory. *Arch Ophthalmol.* 1979;97:1667–72. [PubMed] [Google Scholar]
41. Ritch R, Mudumbai R, Liebmann JM. Combined Exfoliation and Pigment Dispersion:Paradigm of an Overlap Syndrome. *Ophthalmology.* 2000;107:1004–8. [PubMed] [Google Scholar]
42. Prince AM, Ritch R. Clinical Signs of the Pseudoexfoliation Syndrome. *Ophthalmology.* 1986;93:803–7. [PubMed] [Google Scholar]
43. Richardson TM, Hutchinson B, Grant WM. The Outflow Tract in Pigmentary Glaucoma:A Light and Electron Microscopic Study. *Arch Ophthalmol.* 1977;95:1015–25. [PubMed] [Google Scholar]
44. Gottanka J, Johnson DH, Grehn F, Lütjen-Drecoll E. Histologic Findings in Pigment Dispersion Syndrome and Pigmentary Glaucoma. *J Glaucoma.* 2006;15:142–51. [PubMed] [Google Scholar]
45. Dua HS, Dick AD, Watson NJ, Forrester JV. A Spectrum of Clinical Signs in Anterior Uveitis. *Eye (Lond)* 1993;7:68–73. [PubMed] [Google Scholar]
46. Accorinti M, Saturno MC, Paroli MP, De Geronimo D, Gilardi M. Uveitis-Glaucoma-Hyphema Syndrome:Clinical Features and Differential Diagnosis. *Ocul Immunol Inflamm.* 2021;1:1–6. [PubMed] [Google Scholar]

47. Cohen EJ. Iris Ring Melanoma Masquerading as Pigmentary Glaucoma. *Arch Ophthalmol.* 1997;115:1480. [PubMed] [Google Scholar]
48. Richter CU, Richardson TM, Grant WM. Pigmentary Dispersion Syndrome and Pigmentary Glaucoma:A Prospective Study of the Natural History. *Arch Ophthalmol.* 1986;104:211–15. [PubMed] [Google Scholar]
49. Bustamante-Arias A, Ruiz-Lozano RE, Carlos Alvarez-Guzman J, Gonzalez-Godinez S, Rodriguez-Garcia A. Pigment Dispersion Syndrome and its Implications for Glaucoma. *Surv Ophthalmol.* 2021;66:743–760. [PubMed] [Google Scholar]
50. Gandolfi SA, Ungaro N, Tardini MG, Ghirardini S, Carta A, Mora P. A 10-Year Follow-up to Determine the Effect of YAG Laser Iridotomy on the Natural History of Pigment Dispersion Syndrome:A Randomized Clinical Trial. *JAMA Ophthalmol.* 2014;132:1433–8. [PubMed] [Google Scholar]
51. Brusini P, Papa V. Use of Infrared Images of Automatic Refractometer to Screen Pigment Dispersion Syndrome:A Cross-Sectional Observational Study from a Preliminary Hypothesis. *Medical Hypothesis Discovery and Innovation in Optometry.* 2020;1:25–8. [Google Scholar]
52. Lunde MW. Argon Laser Trabeculoplasty in Pigmentary Dispersion Syndrome with Glaucoma. *Am J Ophthalmol.* 1983;96:721–5. [PubMed] [Google Scholar]
53. Ayala M. Long-term Outcomes of Selective Laser Trabeculoplasty (SLT) Treatment in Pigmentary Glaucoma Patients. *J Glaucoma.* 2014;23:616–19. [PubMed] [Google Scholar]
54. Harasymowycz PJ, Papamatheakis DG, Latina M, De Leon M, Lesk MR, Damji KF. Selective Laser Trabeculoplasty (SLT) Complicated by Intraocular Pressure Elevation in Eyes with Heavily Pigmented Trabecular Meshworks. *Am J Ophthalmol.* 2005;139:1110–3. [PubMed] [Google Scholar]
55. Jampel HD. Lack of Effect of Peripheral Laser Iridotomy in Pigment Dispersion Syndrome. *Arch Ophthalmol.* 1993;111:1606. [PubMed] [Google Scholar]
56. Migliazzo CV, Shaffer RN, Nykin R, Magee S. Long-term Analysis of Pigmentary Dispersion Syndrome and Pigmentary Glaucoma. *Ophthalmology.* 1986;93:1528–36. [PubMed] [Google Scholar]
57. Buffault J, Leray B, Bouillot A, Baudouin C, Labbé A. Role of Laser Peripheral Iridotomy in Pigmentary Glaucoma and Pigment Dispersion Syndrome:A Review of the Literature. *J Fr Ophtalmol.* 2017;40:e315–21. [PubMed] [Google Scholar]
58. Akil H, Chopra V, Huang A, Loewen N, Noguchi J, Francis BA. Clinical Results of ab Interno Trabeculotomy using the Trabectome in Patients with Pigmentary Glaucoma Compared to Primary Open Angle Glaucoma. *Clin Exp Ophthalmol.* 2016;44:563–9. [PubMed] [Google Scholar]
59. Wang C, Dang Y, Shah P, Esfandiari H, Hong Y, Loewen RT, et al. Intraocular Pressure Reduction in a Pigmentary Glaucoma Model by Goniotome Ab Interno Trabeculectomy. *PLoS One.* 2020;15:e0231360. [PMC free article] [PubMed] [Google Scholar]
60. Dorairaj SK, Seibold LK, Radcliffe NM, Aref AA, Jimenez-Román J, Lazcano-Gomez GS et al. 12-Month Outcomes of Goniotomy Performed Using the Kahook Dual Blade Combined with Cataract Surgery in Eyes with Medically Treated Glaucoma. *Adv Ther.* 2018;35:1460–9. [PMC free article] [PubMed] [Google Scholar]
61. Brusini P, Papa V. Canaloplasty in Pigmentary Glaucoma:Long-Term Outcomes and Proposal of a New Hypothesis on its Intraocular Pressure Lowering Mechanism. *J Clin Med.* 2020;9:4024. [PMC free article] [PubMed] [Google Scholar]
62. Guerrero-de Ferran C, Rodríguez-García A. Intraocular Pressure Variation During Episodes of Pigment Dispersion and its Relationship with the Development of Pigmentary Glaucoma. *Rev Mex Oftalmol.* 2018;92:8–13. [Google Scholar]