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# PIGMENT DISPERSION SYNDROME WITH ADVANCED GLAUCOMATOUS OPTIC NEUROPATHY: A CASE REPORT

Ophthalomology

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## ABSTRACT

***Background:*** Pigment dispersion syndrome (PDS) is an uncommon cause of secondary open-angle glaucoma, typically presenting in young myopic males. It is characterized by dispersion of iris pigment and its deposition in anterior segment structures, which can result in elevated intraocular pressure (IOP) and glaucomatous optic neuropathy. Although more frequently reported in Caucasians, PDS is rarely described in the Indian population. ***Case Presentation:*** We report a 40-year-old male who presented with a gradual, painless diminution of vision in the right eye for one year. On examination, intraocular pressures were elevated bilaterally (26 mmHg OD, 28 mmHg OS). Slit lamp revealed pigment deposition on the endothelium and anterior lens capsule in both eyes, with iridodonesis and phacodonesis in the right eye. Gonioscopy demonstrated open angles with heavy trabecular pigmentation, and ultrasound biomicroscopy revealed a concave iris configuration in the right eye and a flat iris in the left. Optic nerve evaluation showed advanced glaucomatous optic atrophy (CDR 0.9 OD, 0.8 OS). OCT of the left eye confirmed diffuse RNFL thinning (65 µm), while perimetry demonstrated advanced glaucomatous field loss (MD –26.3 dB). The right eye OCT and perimetry were unreliable due to poor fixation. ***Conclusion:*** This case highlights the importance of recognizing subtle clinical features and structural changes in PDS, even in non-myopic patients from India. Elevated IOP remains the strongest predictor of progression to pigmentary glaucoma. Imaging modalities, such as OCT, UBM, and perimetry, are invaluable in confirming both structural and functional damage. Early recognition, close monitoring, and timely intervention are crucial to prevent irreversible vision loss in these patients.

## KEYWORDS

Pigment Dispersion Syndrome; Pigmentary Glaucoma; Ultrasound Biomicroscopy; Optical Coherence Tomography; Perimetry; Case Report

## INTRODUCTION

Pigment dispersion syndrome (PDS) is characterized by the liberation of pigment granules from the posterior iris pigment epithelium, depositing on anterior segment structures. The classic diagnostic triad includes Krukenberg's spindle, mid-peripheral iris transillumination defects, and trabecular meshwork pigmentation (1,2). PDS typically presents in the third to fourth decades and frequently affects myopic males (3,5,10). Importantly, cases have been reported after refractive surgery and phakic intraocular lens implantation, emphasizing the role of iris–lens–zonular dynamics in pigment liberation (8,9).

Although PDS is more commonly described in Caucasians, it is rarely reported in Indian populations (3,10). A significant proportion of patients eventually progress to pigmentary glaucoma (PG), with the strongest predictor being elevated baseline intraocular pressure (IOP) (4).

We present a case of PDS with advanced glaucomatous optic neuropathy in a 40-year-old Indian male, emphasizing clinical features and the role of OCT, UBM, and perimetry in documenting structural and functional damage.

## CASE REPORT

### History

A 40-year-old Hindu male presented to M & J Eye Institute with a chief complaint of gradual, painless diminution of vision in the right eye for one year. The patient was asymptomatic before onset and denied a history of ocular trauma, glare, halos, photophobia, redness, or systemic illness.

### Examination

- Visual acuity: Right eye – perception of light with unreliable projection of rays; Left eye – 6/6 with –0.5 DCyl at 15°.
- Intraocular pressure (Goldmann applanation): 26 mmHg (OD), 28 mmHg (OS).
- Central corneal thickness: 536 µm (OD), 528 µm (OS).
- Slit lamp examination: Right eye – deep anterior chamber, iridodonesis, phacodonesis, diffuse pigment deposition on corneal endothelium and anterior lens capsule, sluggish pupil, clear lens. Left eye – grossly normal anterior segment except for scattered pigment on endothelium and lens capsule.
- Gonioscopy: Right eye – Shaffer grade 3 open angles, heavily pigmented trabecular meshwork, Schwalbe's line visible, markedly concave iris configuration. Left eye – Shaffer grade 3 open angles, pigmented trabecular meshwork, flat-to-mildly concave iris.
- Fundus examination (78D): Right eye – cup-to-disc ratio 0.9, deep cup, vessel bayonetting, laminar dot sign, advanced glaucomatous optic atrophy. Left eye – cup-to-disc ratio 0.8, deep cup, bayonetting, laminar dot sign, pale neuroretinal rim.
- Peripheral retina (indirect ophthalmoscopy): Normal in both eyes.

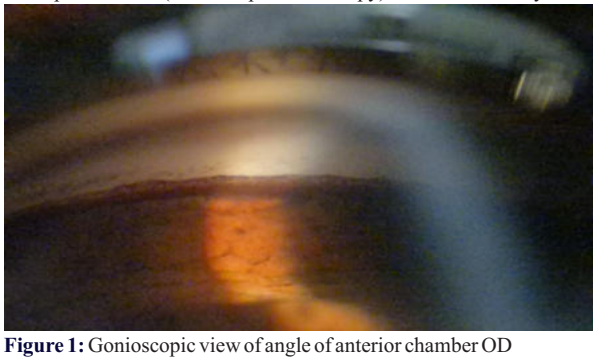

**Figure 1:** Gonioscopic view of angle of anterior chamber OD

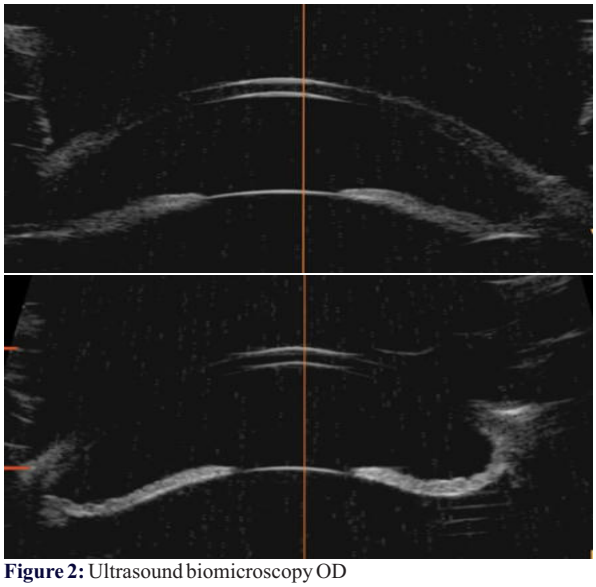

**Figure 2:** Ultrasound biomicroscopy OD

**Investigations**

● Optical coherence tomography (OCT):
○ Right eye OCT was unreliable due to poor fixation.
○ Left eye OCT showed diffuse RNFL thinning with average thickness of 65 µm, rim area 0.443 mm², vertical cup–disc ratio 0.85, and increased cup volume 1.53 mm³.

● Ultrasound biomicroscopy (UBM):
○ Right eye – concave iris configuration, anterior chamber depth (ACD) 3.54 mm, lens thickness 3.98 mm.
○ Left eye – flat iris configuration, ACD 3.39 mm, lens thickness 3.98 mm.

● Perimetry (Octopus static perimetry):
○ Right eye – unreliable due to poor vision.
○ Left eye – dense inferior arcuate scotoma extending into fixation with scattered superior defects. Mean sensitivity (MS) 18.4 dB, mean deviation (MD) –26.3 dB, square loss variance (sLV) 2.9 dB. Findings consistent with advanced glaucomatous visual field loss.

**Table 1: Summary Of Clinical And Imaging Findings**

| Parameter | Right Eye (OD) | Left Eye (OS) |
|---|---|---|
| Visual acuity | Perception of light, unreliable projection of rays | 6/6 with –0.5 DCyl at 15° |
| Intraocular pressure | 26 mmHg | 28 mmHg |
| Central corneal thickness | 536 µm | 528 µm |
| Slit lamp examination | Deep anterior chamber, iridodonesis, phacodonesis, diffuse endothelial & lens capsule pigment, sluggish pupil, clear lens | Normal anterior segment, few endothelial and anterior capsule pigment |
| Gonioscopy | Shaffer grade 3 open angles, heavily pigmented TM, Schwalbe's line visible, concave iris | Shaffer grade 3 open angles, pigmented TM, flat iris configuration |
| Optic nerve head (78D) | CDR 0.9, advanced glaucomatous optic atrophy with deep cup, vessel bayonetting, laminar dot sign | CDR 0.8, deep cup, bayonetting, laminar dot sign, pale neuroretinal rim |
| Retina (IO) | No peripheral abnormalities | No peripheral abnormalities |
| OCT (RNFL analysis) | Unreliable due to poor fixation | Average RNFL 65 µm, rim area 0.443 mm², vertical CDR 0.85, diffuse RNFL loss |
| UBM | Concave iris, ACD 3.54 mm, LT 3.98 mm | Flat iris, ACD 3.39 mm, LT 3.98 mm |
| Perimetry | Unreliable due to poor vision | Dense inferior arcuate scotoma into fixation, MD –26.3 dB, advanced field loss |

## DISCUSSION

This patient illustrates hallmark features of PDS with secondary ocular hypertension and asymmetric glaucomatous damage. The combination of heavy trabecular pigmentation, fine endothelial pigment, and concave iris configuration strongly supports the reverse pupillary block hypothesis, wherein posterior iris bowing increases irido-zonular contact and pigment release (5).

Risk of progression: Long-term studies show a 10% conversion from PDS to PG at 5 years and 15% at 15 years, with baseline IOP >21 mmHg being the strongest predictor of progression (3,4). Our patient presented with IOPs of 26 and 28 mmHg, consistent with high-risk disease.

Anterior segment configuration: UBM demonstrated a concave iris in the right eye, consistent with earlier studies that showed UBM as critical for documenting iris contour in PDS/PG (6,7). The asymmetry between eyes may explain the greater pigment dispersion and optic nerve damage in OD.

Functional assessment: Perimetry confirmed advanced glaucomatous loss in OS, correlating with OCT and optic disc findings. This aligns with prior studies showing structural-functional concordance in advanced PDS-related glaucoma (10).

Differential and associations: PDS has also been reported following refractive surgery and phakic intraocular lens implantation, where anterior segment alterations precipitate pigment dispersion (8,9). In our patient, the absence of a surgical history suggests de novo PDS.

Management: Management parallels primary open-angle glaucoma: topical therapy to lower IOP remains first-line, while miotics may reduce iris concavity. Nd:YAG laser peripheral iridotomy and argon laser trabeculoplasty have shown mixed results (11,12). Given advanced optic nerve damage, surgical intervention may be considered. Lifelong surveillance with OCT, UBM, and perimetry is essential.

## CONCLUSION

Pigment dispersion syndrome, though rare in Indian patients, should be suspected in cases of elevated IOP with subtle pigment deposition in the anterior segment. This case demonstrates bilateral PDS with asymmetric iris configuration on UBM and advanced glaucomatous optic neuropathy, functionally confirmed by perimetry. Elevated IOP remains the key predictor of conversion to PG (3,4,10). Imaging modalities such as OCT and UBM provide objective insights into structural damage and iris anatomy, while perimetry demonstrates functional impairment. Early recognition and vigilant follow-up are vital to prevent irreversible vision loss.

**List of Abbreviations**

● PDS: Pigment Dispersion Syndrome
● PG: Pigmentary Glaucoma
● IOP: Intraocular Pressure
● OCT: Optical Coherence Tomography
● UBM: Ultrasound Biomicroscopy
● RNFL: Retinal Nerve Fiber Layer
● MD: Mean Deviation
● MS: Mean Sensitivity
● sLV: Square Loss Variance
● ACD: Anterior Chamber Depth
● LT: Lens Thickness
● CDR: Cup-to-Disc Ratio
● OD: Oculus Dexter (Right Eye)
● OS: Oculus Sinister (Left Eye)
● PL: Perception of Light
●DCyl: Diopter Cylinder
● TM: Trabecular Meshwork

**Declarations**

**● Ethics approval and consent to participate**
Institutional ethics approval was not required for this single-patient case report, in accordance with local regulations. Written informed consent was obtained from the patient to participate in this study.

**● Consent for publication**
Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical details and images. A copy of the consent is available with the corresponding author.

**● Availability of data and materials**
All clinical data supporting the findings of this case report are available from the corresponding author upon reasonable request.

**● Competing interests**
The author declares no competing interests.

**● Funding**
No funding was received for this work.

**● Authors' contributions**
Dr. Anshumalee Patel was responsible for the conception, clinical examination, acquisition and interpretation of data, and drafting of the manuscript. The author approved the final version of the manuscript.

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