When Inflammation Masks Anatomy: Bilateral Acute Uveitis Revealing Plateau Iris Syndrome with Severe Ocular Hypertension

Abstract

Background:
Ocular hypertension in the context of acute uveitis is a common but complex condition, often attributed to inflammatory mechanisms. However, underlying anatomical abnormalities such as plateau iris syndrome may coexist and complicate both diagnosis and management. The association between acute uveitis and plateau iris syndrome is exceptionally rare.

Case presentation:
We report the case of a 50-year-old man with a history of ocular hypertension who presented with bilateral acute anterior uveitis associated with severe ocular hypertension (48 mmHg). Clinical examination revealed marked anterior segment inflammation with granulomatous keratic precipitates, severe anterior chamber reaction, and extensive posterior synechiae. Fundus examination showed advanced glaucomatous optic neuropathy. Fluorescein angiography demonstrated peripheral retinal periphlebitis and optic disc leakage. Optical coherence tomography confirmed severe nerve fiber layer loss. Ultrasound biomicroscopy revealed bilateral plateau iris configuration, which had not been previously diagnosed.

Management and outcome:
Despite maximal hypotensive therapy, intraocular pressure remained elevated. Following initiation of high-dose intravenous corticosteroids, intraocular pressure normalized rapidly, highlighting the predominant role of inflammation.

Conclusion:
This case underscores the importance of identifying underlying anatomical factors in uveitic ocular hypertension. However, controlling inflammation remains the primary therapeutic priority. Plateau iris syndrome may act as an aggravating factor but should not divert attention from the inflammatory mechanism.

Keywords:
Uveitis; Plateau iris syndrome; Ocular hypertension; Corticosteroids; Diagnostic challenge

Introduction

Ocular hypertension in acute uveitis is a frequent and potentially vision-threatening complication. Its pathophysiology is multifactorial, involving inflammatory obstruction of the trabecular meshwork, increased aqueous humor viscosity, and structural damage to outflow pathways.

Plateau iris syndrome is a rare anatomical condition characterized by an anterior positioning of the ciliary body, leading to mechanical angle closure despite a normal central anterior chamber depth. It is typically diagnosed using ultrasound biomicroscopy (UBM).

The coexistence of inflammatory and anatomical mechanisms in ocular hypertension represents a significant diagnostic and therapeutic challenge. While uveitis-related hypertonia is usually managed with anti-inflammatory therapy, plateau iris may lead clinicians to prioritize hypotensive treatment.

We report a rare case of bilateral acute uveitis associated with plateau iris syndrome, highlighting the importance of a pathophysiology-driven approach.

Case Report

A 50-year-old man presented with bilateral ocular redness, pain, and decreased vision evolving over several days.

Medicalhistory

- Previous episode of ocular hypertension (2015), treated with topical therapy
- Recurrence of elevated intraocular pressure in 2020 (54 mmHg OD, 47 mmHg OS)
- Familyhistory:
    - Behçet'sdisease (brother)
    - Primary open-angle glaucoma (father and sister)

ClinicalExamination

- **Visual acuity:** 7/10 in botheyes
- **Intraocular pressure:** 48 mmHg bilaterally (under treatment)

Anterior segment findings

- Eyelidedema and hyperemia
- Conjunctivalchemosiswithsubconjunctivalhemorrhage
- Diffuse keratic precipitates, including large granulomatous precipitates
- Severe anterior chamber inflammation (Tyndall 4+)
- Inflammatory membrane formation
- Posteriorsynechiae

Fundus examination

- No vitritis or posterior inflammation
- Optic disc:
    - Right eye: almost total cupping (0.9)
    - Lefteye: advancedcupping (0.8)
- Retinaotherwiseunremarkable

Ophthalmologic Investigations

FluoresceinAngiography

- Peripheralretinalperiphlebitis
- Bilateraloptic disc leakage

Optical CoherenceTomography (OCT)

- Severe RNFL and GCC loss in the right eye
- Advanced glaucomatous damage
- Early involvement in the left eye

Visual Field

- Right eye: tubular vision
- Left eye: early visual field defects

UltrasoundBiomicroscopy (UBM)

- Bilateral plateau iris configuration
- Anterior positioning of the ciliary body
- Narrow iridocorneal angle

EtiologicalWorkup

Neurologicalevaluation

- Normal clinicalexamination
- Brain MRI: normal

Lumbarpuncture

- Normal

Inflammatory and autoimmune tests

- ANA: negative
- HLA-B27, HLA-B51: negative
- ACE: normal
- CRP: negative

Infectiousworkup

- HSV serology: negative
- Syphilis (TPHA/VDRL): negative
- Tuberculosis (Quantiferon): indeterminate

Management

The patient was hospitalized for urgent management.

Anti-inflammatorytreatment

- Intravenous methylprednisolone (1 g/day for 3 days)
- Oral corticosteroid relay (1 mg/kg/day)
- Topicalcorticosteroids
- Cycloplegic (atropine)

Intraocular pressure management

- Mannitol infusion
- Oral acetazolamide
- Topical hypotensive therapy (triple therapy)

Outcome

- Day 1: persistent ocular hypertension despite maximal therapy
- Day 2: rapid normalization of intraocular pressure following IV corticosteroids
- Day 5:
    - IOP stabilized at 18–20 mmHg

- Decrease in inflammation
- Stable visualacuity

Discussion

This case illustrates a rare and complex association between acute uveitis and plateau iris syndrome.

Plateau iris is typically responsible for mechanical angle closure due to anterior displacement of the ciliary body. However, in this case, the dramatic response of intraocular pressure to corticosteroid therapy strongly indicates that inflammation was the primary driver of hypertonia.

Inflammatorymechanismsinclude:

- Trabecularmeshworkedema
- Cellular debris obstruction
- Increasedaqueoushumorviscosity

The presence of plateau iris likely contributed to angle narrowing, acting as an aggravating factor rather than the primary cause.

This highlights a key clinical message:

Inuveitic ocular hypertension, inflammation must be treated first—even in the presence of anatomical predisposition.

Failure to recognize this may lead to inappropriate escalation of hypotensive therapy without addressing the underlying mechanism.

To our knowledge, the association between acute uveitis and plateau iris syndrome has not been previously reported, making this case particularly noteworthy.

Conclusion

This case emphasizes the importance of a comprehensive diagnostic approach in uveitic ocular hypertension.

While plateau iris syndrome may contribute to angle narrowing, inflammation remains the primary therapeutic target. Early and aggressive corticosteroid therapy is essential to control intraocular pressure and prevent irreversible optic nerve damage.

Clinicians should avoid focusing solely on intraocular pressure and instead adopt a pathophysiology-based management strategy.

References

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