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*RESEARCH ARTICLE*

# WHEN INFLAMMATION MASKS ANATOMY: BILATERAL ACUTE UVEITIS REVEALING PLATEAU IRIS SYNDROME WITH SEVERE OCULAR HYPERTENSION

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

## *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 rare and has been only scarcely reported in the literature.

**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 reacti on, 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 on day 1. A rapid decrease in intraocular pressure was observed within 24 hours after initiation of high-dose intravenous corticosteroids, reaching 18–20 mmHg by day 5, with concomitant resolution of inflammation.

**Conclusion:** This case suggests that, in uveitic ocular hypertension, inflammation may represent the predominant mechanism even in the presence of anatomical abnormalities such as plateau iris syndrome. Plateau iris may act as an aggravating factor rather than the primary cause. These findings should be interpreted cautiously given the single-case design.



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## 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.Several recent studies have emphasized the central role of inflammation in uveitic glaucoma, highlighting the importance of early anti-inflammatory therapy.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).Its pathophysiology involves anterior rotation of the ciliary processes, resulting in a narrow angle configuration independent of pupillary block.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.This diagnostic ambiguity may result in inappropriate management strategies if the predominant mechanism is not correctly identified.We report a rare clinical observation of bilateral acute uveitis associated with plateau iris syndrome, highlighting the importance of a pathophysiology-driven approach.

**Ethical approval and consent:-**

Written informed consent was obtained from the patient for publication of this case report. The study adhered to the principles of the Declaration of Helsinki.

**Case Report:-**

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

**Medical history:-**

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

This family history was carefully considered but no clinical or biological evidence supported a diagnosis of Behçet's disease in the patient.

**Clinical Examination:-**

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

**Anterior segment findings:-**

- Eyelid edema and hyperemia
- Conjunctival chemosis with subconjunctival hemorrhage
- Diffuse keratic precipitates, including large granulomatous precipitates
- Severe anterior chamber inflammation (Tyndall 4+)
- Inflammatory membrane formation
- Posterior synechiae

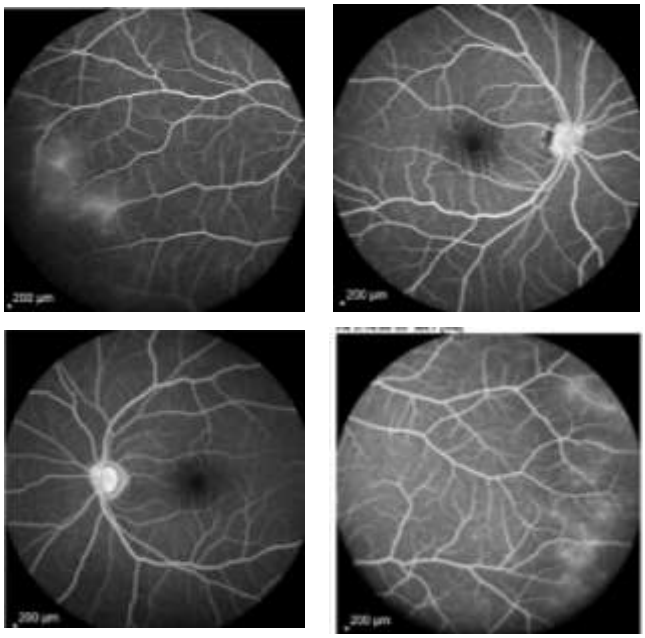
**Fundus examination:-**

- No vitritis or posterior inflammation
- Optic disc:
  - Right eye: almost total cupping (0.9)
  - Left eye: advanced cupping (0.8)
- Retina otherwise unremarkable

Baseline optic nerve status prior to this episode was not available, representing a limitation in distinguishing chronic from acute glaucomatous damage.

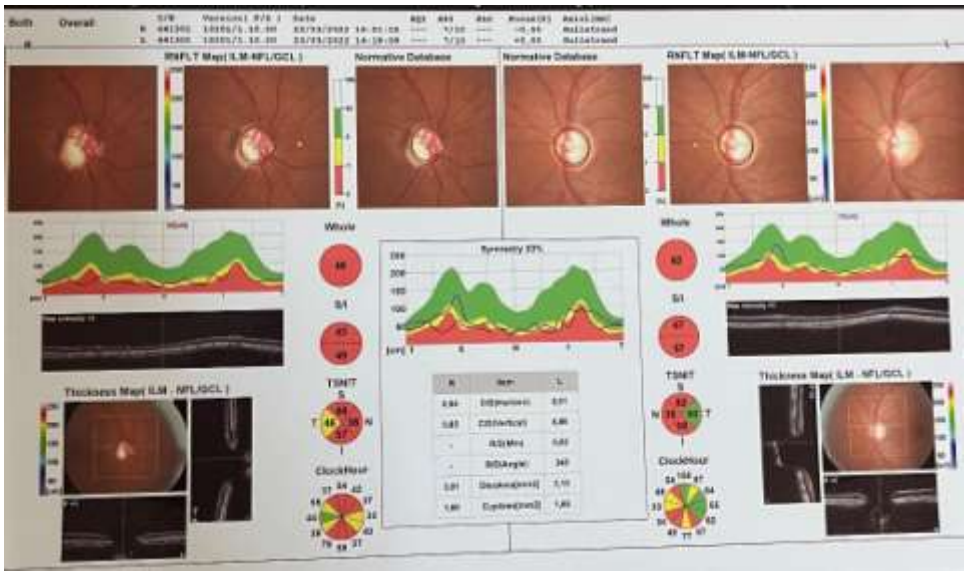
**Ophthalmologic Investigations:-**
**Fluorescein Angiography:-**

- Peripheral retinal periphlebitis
- Bilateral optic disc leakage



**Optical Coherence Tomography (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

**Ultrasound Biomicroscopy (UBM):-**

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

**Etiological Workup:-**

**Neurological evaluation:-**

- Normal clinical examination
- Brain MRI: normal

**Lumbar puncture:-**

- Normal

**Inflammatory and autoimmune tests:-**

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

**Infectious workup:-**

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

The indeterminate Quantiferon result was considered non-contributory in the absence of clinical or radiological evidence of tuberculosis.

**Management:-**

The patient was hospitalized for urgent management.

**Anti-inflammatory treatment:-**

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

**Intraocular pressure management:-**

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

Treatment decisions were based on standard management guidelines for uveitic glaucoma, prioritizing inflammation control.

**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 visual acuity

Long-term follow-up data were not available, which represents a limitation of this report.

## Discussion:-

This case illustrates a complex and uncommon 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.

**Inflammatory mechanisms include:**

- Trabecular meshwork edema
- Cellular debris obstruction
- Increased aqueous humor viscosity

These mechanisms are well described in uveitic glaucoma and supported by previous studies.
The presence of plateau iris likely contributed to angle narrowing, acting as an aggravating factor rather than the primary cause.However, the exact contribution of plateau iris cannot be definitively established in this single case.

**Differential diagnoses should also be considered, including:**

- Herpeticuveitis
- Posner–Schlossman syndrome
- Secondary angle-closureglaucoma
- Idiopathicuveitis

These conditions were excluded based on clinical presentation, laboratory investigations, and imaging findings.

**This highlights a key clinical message:**

☞ In uveitic ocular hypertension, inflammation should be treated as the primary mechanism before escalating hypotensive therapy, even in the presence of anatomical predisposition.
Failure to recognize this may lead to inappropriate management strategies.
Although this association appears uncommon, similar cases have been sparsely described in the literature, and further studies are needed to better characterize this entity.

## Limitations:-

This report is limited by its single-case design, absence of long-term follow-up, and lack of quantitative imaging data and illustrative figures. These limitations reduce the generalizability of the findings.

## 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.
However, conclusions should remain cautious given the single-case nature of this report, and further studies are required to confirm these observations.

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