Branch Retinal Occlusion (BRAO) refers to decreased arterial blood flow of the retina leading to downstream ischemic damage to the inner retinal layers. Retinal emboli are visible on fundoscopic exam in 62% of BRAO cases.

Emboli commonly composed of cholesterol (Hollenhorst plaque), platelet-fibrin clot or calcium plaques. Risk factors include hypertension, carotid stenosis, history of stroke or TIA and hypercholesterolemia.

Purpose

- Branch retinal artery occlusion is a common disorder of the ocular vasculature, which stems from the occlusion of a branch of the central retinal artery.
- Symptoms of BRAO include sudden, painless, severe vision loss or visual field deficit, usually unilaterally.
- This is a case report of a patient who experienced visual field deficits from an old stroke in addition to a branch retinal occlusion.

Background

76-year-old male presents to the glaucoma clinic with an acute onset of painless monocular visual field loss OS. He stated that he experienced visual field deficits unilaterally for over a year.

Chief complaint:
- Abrupt painless vision loss OS 10 days ago
- 50% superior field vision loss OS

Past Ocular History:
- Mild POAG OU, Cataracts OU
- Hx of stroke with subsequent visual field defect, subtotal left inferior homonymous quadrantanopia (~4 years ago)

Past Medical History:
- Stroke, Hypertension, Hypercholesterolemia

Ocular Medications:
- Latanoprost 0.005% OU nightly
- Aspirin 81mg once daily

Examination:
- BCVA: OD: cc 20/60, PH 20/40
- OS: cc 20/30, PH 20/20
- iCare: OD: 12; OS: 12
- Pupils were equal, round and reactive to light
- SLE was remarkable for dermatomalaskis, cataracts OU
- DFE was remarkable for Hollenhorst plaque along inferior arcade OS

Imaging:
- Fundus photos (Image 1 & 2)
- OCT
- FFA

Case Report

Branch Retinal Artery Occlusion

Acute onset of painless monocular vision impairment

Signs:
- Embolus visible on fundoscopic exam
- Retinal ischemia (cotton wool spots and retinal whitening)
- Non-perfusion of vessels on fluorescein angiography
- Retinal edema and atrophy (can be visualized with SD-OCT)

Systemic Workup:
- Carotid doppler ultrasound, cardiac echocardiography, brain imaging (CT or MR angiography)

Treatments:
- No known treatments for vision recovery
- Antiplatelet therapy (TPA) may be initiated within four hours of symptom onset

Long-term stroke prevention:
- Statin, aspirin 81mg, clopidogrel (DAPT)

Discussion

Branch Retinal Artery Occlusion

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References


Initial Assessment

Management: Prompting systemic workup
- Carotid artery Doppler ultrasound reading
  - Right: Less than 50% right internal carotid artery stenosis
  - Left: 50-79% stenosis in the left internal carotid artery, with presence of a plaque

CT Angiogram
- Decreased vessel lumen of the right and left carotid arteries

Treatment: Carotid Endarterectomy
- The most pertinent cause of concern is ruling out or treating a stroke in the brain

Following up:
- Routine ophthalmic examination
- Continuation on vaso-occlusive disease prophylaxis therapy (statin, baby aspirin, smoking cessation and diet control)

Conclusion

In similar cases of artery occlusion, goal is to identify etiology of embolism and then to take secondary prevention measures to decrease the likelihood of subsequent strokes and other ocular ischemic events.

- Refer patient out to immediate complete vascular workup
- Continue working with primary care physician and vascular surgeon to manage patient’s vascular/stroke risk factors

Disclosures and Funding

- No relevant financial disclosures.
**DIFFERENTIAL DIAGNOSIS**

- Pituitary Adenoma:
  - Tumor of the pituitary gland leading to compression of optic chiasm superior to gland
  - Bilateral pituitary adenopathy:
    - Outgrowth of remnant of Rathke’s pouch near sella
- Meningioma:
  - Neoplasm of arachnoid cells that may be in parasellar region
- Anterior Communicating Artery Aneurysm:
  - Located superior to optic chiasm leading to impingement of optic nerve fibers at chiasm
- Tilted Disc Syndrome:
  - Oblique insertion of optic nerve with axonal dysgenesis of nasal nerve fibers

**DIAGNOSIS**

- Urgency of lab testing and imaging is based on severity of neurological or systemic symptoms.
- MRI of brain and orbits with and without contrast with attention to sella.
- Blood serum levels of: Cortisol, FSH, LH, Prolactin, TSH, Free T4, ACTH, GH

Based on the MRI findings the patient was diagnosed with a bitemporal hemianopsia secondary to non-functioning pituitary macroadenoma.

**DISCUSSION**

Pituitary adenomas comprise 10-15% of all intracranial masses. Adenomas are classified based on size and hormone secretion. Adenomas less than 10mm in size are categorized as a microadenoma, those larger are macroadenomas. Those that do not cause elevated levels of hormones detectable in blood or produce clinical manifestations are classified as non-functioning (28-37%). The location of the pituitary adenoma inside the sella turcica forces the adenoma superiorly resulting in compression of the optic nerve and chiasm. The most common hormone hypersecretions disorders are hyperprolactinemia, acromegaly, and Cushings disease. Fatigue, loss of libido, erectile dysfunction, oligomenorrhea or amenorrhea are common clinical presentations. Headaches and visual changes are the most common neurological symptoms. The greatest concern is pituitary apoplexy.

**CONCLUSION**

Visual symptoms are more common in non-function macroadenomas. Serum prolactin greater than 250 mcg/L is suggestive of prolactinoma with symptoms such as decreased libido, impotence, oligomenorrhea or amenorrhea. Treatment of tumors include transsphenoidal microsurgery, radiation and medical therapy. A follow-up every 3 months after treatment is recommended to assess changes to visual acuity or visual field. Optic nerve pallor is the best predictor of visual prognosis. Facial amslers may be more sensitive than confrontation fields. After treatment, visual acuity can recover faster than visual field defect.

**REFERENCES**

[1] Two Halves Make a Whole: Management of Pituitary Macroadenoma
Jorge Giraldo, OD, Kasey Zann, OD
Miami VA Medical Center, Miami, FL

- **CASE HISTORY**
  - A 47-year-old Hispanic male presents to the Miami VA eye clinic for his yearly comprehensive eye exam. The patient reports central to temporal blurry vision of the left eye that started four months ago and became worse after a COVID-19 infection one month prior. During refraction the patient was reading only the last letter of each line for the left eye. A facial amslers confirmed that the central to temporal vision of the left eye was blurry with similar complaints of the right eye starting farther temporally. A 30-2 visual field demonstrated bitemporal visual field defects. Ganglion cell analysis showed diffuse atrophy of both eyes. An MRI of the brain and orbits with attention to sella was ordered and showed a lesion consistent with a pituitary macroadenoma. Due to complaints of worsening central vision, the pituitary macroadenoma was removed by transsphenoidal resection. At the patient’s follow-up, visual acuity and visual field improved.

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Two May Not Always Be Better Than One

Dr. Timothy Shoff OD, Dr. Joshua Pasol MD, Dr. Kasey Zann OD

CASE HISTORY

A 63-year-old white male presents to the neuro-ophthalmology clinic with a referral from the emergency department for bilateral papilledema. He reports that he noticed a black spot in his central vision that does not move like a typical floater in his right eye. He also reported visual disturbances in his peripheral vision that reminded him of Christmas tree lights when he focuses on objects. He denied pain on eye movement, sculp tenderness, jaw pain, loss of appetite, or unintentional weight loss. His past medical history is positive for diabetes, hypertension, hyperlipidemia, gout, and chronic kidney disease. His current medications include Alogliptin, Aspirin, Atorvastatin, Furosemide, Loratadine, Metoprolol, Tamsulosin, and Vitamin D.

INITIAL PRESENTATION

TREATMENT CONSIDERATIONS

Treatment in NAAION is relatively controversial. Multiple studies have investigated different treatments. Optic nerve decompression surgery has been determined to cause more harm than good. Oral steroids have been one of the most studied treatment options. A Complete Blood Count (CBC), C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR) were ordered for same-day testing. In addition, an MRI, MRV, Temporal Artery Biopsy (TABA), and Lumbar Puncture (LP) were ordered. The CBC, TBA, MRV, and Lumbar Puncture results were all within normal limits. The CRP (21) and ESR (45) came back slightly elevated, which could be contributed by his diabetes, hypertension, and renal disease. The patient was started on 30mg/day x 1 week of oral prednisolone with a taper schedule of 20mg/day x 1week, then 10mg/day until his 1-month follow-up. At the 1-month follow-up, the patient reported no changes in visual symptoms, and all preliminary testing remained stable. The visual field and OCT showed improvement, and he was instructed to continue 10mg/day of prednisolone for three more weeks. The patient’s blood work was repeated and returned within normal limits. At the 3-month mark, the patient reported stable visual symptoms, but the CRP remained elevated, and the prednisolone was tapered and stopped. Our patient was referred for a sleep study and to the low vision clinic. He was scheduled to return to the neuro-ophthalmology clinic in 3 months. The patient received a complete contrast evaluation at the low vision clinic, where a yellow filter improved his low contrast symptoms, and the sleep study results are still pending.

CONCLUSION

Nonarteritic Anterior Ischemic Optic Neuropathy is the most common cause of acute optic neuropathy in individuals over 50 years old. Patients with nerve edema, peripapillary hemorrhaging, and vision reduction need an immediate workup to rule out severe life-threatening diseases. Even though there is no proven effective treatment for patients with NAAION, it is essential to identify and manage any underlying conditions and risk factors that increase the likelihood of NAAION in the fellow eye. In our patient’s case, he had multiple risk factors for his simultaneous presentation, including diabetes, hypertension, hyperlipidemia, gout, chronic kidney disease, and he was taking metoprolol.

REFERENCES

LEARNING OBJECTIVES

1. Review the presentation of ocular graft-versus-host-disease.
2. Be familiarized with diagnosis including correlation of patient’s symptoms and history to examination findings
3. Outline the pathophysiology and differences in management and treatment strategies of ocular graft-versus-host-disease

CASE PRESENTATION

A 75-year-old male with a past medical history of non-Hodgkin’s lymphoma (10 years ago) presents to the hospital eye clinic, complaining of blurriness and irritation OD>OS

HPI:
- Progressively worsening blur and then irritation OD>OS onset 10 days ago
- Primary eye exam two days ago, and was referred for management of chronic dry eye
- Came to hospital eye clinic after onset for a general evaluation and to establish care, recently moved from Virginia
- Long standing history of associated pain and blurry vision
- No visual improvement since onset
CASE PRESENTATION

Past Ocular History:
- Dry Eye Syndrome OU
- Presbyopia OU, wears glasses
- Cataracts OU

Past/Pertinent Medical History:
- Non-Hodgkin lymphoma
  - Treated with a hematopoietic stem cell transplant ten years prior
- Hypertension
- Hypercholesterolemia
- Current daily smoker, one pack per day

CASE PRESENTATION

Ocular Medications:
- Refresh Artificial Tears as needed
- Refresh Gel every night

Pertinent Systemic Medications:
- Lisinopril 10mg once daily
- Aspirin 81mg once daily
- Atorvastatin 20mg once daily

No significant past trauma, allergies, or other contributory family history.

CASE PRESENTATION: EXAM

BCVA
OD: cc 20/50, PH 20/NI
OS: cc 20/25, PH 20/NI

Refraction
OD: +2.50-0.50x180 20/50
OS: +2.25-0.25x180 20/25

Pupils
PERRL, 4mm->2mm OU, no APD

Confrontation Visual Field
Full to Finger Count OD/OS

IOPs (Goldmann)
OD: 14
OS: 12

Adnexa: 1+ MGD OU
Conjunctiva/Sclera: 1+ diffuse injection OU
Cornea: punctate epithelial erosions centrally and inferiorly OD, inferiorly OS; (-)infiltrates/edema
Anterior Chamber: deep and quiet OU; (-) cells/flare
Iris: flat and intact OU
CASE PRESENTATION: EXAM

Lens: 1+ nuclear sclerosis OU
Vitreous: syneresis OU
Optic Nerve: 0.3/0.25 pink and distinct OD/OS
Vessels: 2/3 with slight attenuation OU
Macula: flat and intact OU
Periphery: flat and intact OU

ADDITIONAL TESTING

Corneal staining:
Punctate epithelial erosions were noted on the central and inferior cornea of the right eye and inferior cornea of the left eye

Inflammady
Detects matrix metalloproteinase 9
- Measuring severe ocular surface inflammation

ADDITIONAL TESTING

Schirmer 1 Test
Wetting of paper strip after 5 minutes
- Reduced tear production
CASE PRESENTATION

Diagnosis:
Inflammatory Dry Eye Syndrome secondary to Ocular Graft-Versus-Host Disease

…prompting additional treatment

GRAFT-VERSUS-HOST-DISEASE (GVHD) OVERVIEW

- Common complication following allogeneic hematopoetic stem cell transplantation
- Abnormal immune response to healthy host tissue
- Characterized as acute or chronic
- Currently defined on specific tissue involvement
- Ocular manifestations more common in chronic form
- ~50% of individuals after allogeneic bone marrow transplant develop ocular complications

OCULAR GRAFT-VERSUS-HOST-DISEASE (GVHD) OVERVIEW

- Involves T-cell mediated infiltration and destruction of tear producing glands
- Most common clinical manifestations include keratoconjunctivitis sicca and cicatricial conjunctivitis
- Major cause of long-term morbidity in GVHD
- Typically does not lead to permanent visual loss

DRY EYE SYNDROME

The most frequent complication of ocular GVHD

- Symptoms:
  - dryness
  - burning
  - pain
  - redness
  - light sensitivity
  - fluctuating or blurred vision
- Signs:
  - Meibomian gland obstruction
  - Decreased tear production
  - Rapid tear film break up time (TBUT)
  - Punctate epithelial keratopathy
FURTHER COMPLICATIONS OF GVHD

- Corneal ulceration
- Corneal perforation
- Filamentary keratitis
- Conjunctival scarring
- Cataract formation

TESTING

- Schirmer testing
- Ocular Surface Disease Index (OSDI) or Dry Eye Questionaire-5 (DEQ-5)
- Fluorescein corneal staining
- Tear film breakup time

GRADING OCULAR SYMPTOMS OF GVHD

<table>
<thead>
<tr>
<th>Score</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No symptoms</td>
</tr>
<tr>
<td>1</td>
<td>Mild dry eye symptoms not affecting daily activities (requiring eye drops &lt;3x per day) or asymptomatic signs of KCS</td>
</tr>
<tr>
<td>2</td>
<td>Moderate dry eye symptoms partially affecting daily activities (requiring drops &gt;3x per day or punctal plugs) without vision impairment</td>
</tr>
<tr>
<td>3</td>
<td>Severe dry eye symptoms significantly daily activities (special eyewear to relieve pain) or unable to work because of ocular symptoms or loss of vision caused by KCS</td>
</tr>
</tbody>
</table>

OCULAR GVHD DIAGNOSTIC CRITERIA AND GRADING SCALE
OCULAR GVHD DIAGNOSTIC CRITERIA AND GRADING SCALE

ACUTE GVHD GRADING CRITERIA

CHRONIC GVHD GRADING CRITERIA

TREATMENT OPTIONS

- Preservative-free artificial tears
- Lubricating viscous ointment
- Autologous serum eyedrops
- Oral doxycycline
- Topical corticosteroids
- Topical immunosuppressants
- Moisture goggles
MORE TREATMENT OPTIONS

- Punctal plugs
- Partial tarsorrhaphy
- Amniotic membrane transplantation
- Deep anterior lamellar keratoplasty
- Penetrating keratoplasty

TREATMENT SUMMARY

<table>
<thead>
<tr>
<th>Level</th>
<th>Recommended measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Unpreserved tears, gel, ointment. Sodium Cytosporin A. Steroids. Nutritional supplements.</td>
</tr>
</tbody>
</table>

FUTURE DIAGNOSTICS

- Meibography
- Tear Interferometry
- Tear Film Osmolarity

FUTURE TREATMENT

Contact lenses (PROSE)
CONCLUSION

- Ocular GVHD is a complex and challenging condition to diagnose and manage
- Early diagnosis is essential to reduce or even prevent severe complications
- Multidisciplinary approach is important to determine when systemic, topical or other therapy options are best

SPECIAL THANKS TO:
DIVY MEHRA OMS-IV
NIRMANI KARUNATHILAKE OD
JEFFERY CURRY OD, FAAO

REFERENCES


QUESTIONS?

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321-652-2326

Lamellar Hole-Associated Epiretinal Proliferation
Charateristics & Potential Implications on Prognosis & Management of Lamellar Macular Hole

Ashley Ireland, O.D.
Resident in Primary Care with an Emphasis in Ocular Disease
Nova Southeastern University

Learning Objectives

1. Review normal vitreous physiology and interaction with the retina.
2. Contrast the physiologic versus pathologic vitreous aging process.
3. List the spectrum of vitreomacular interface (VMI) disorders that can arise from anomalous posterior vitreous detachment.
4. Understand the pathogenesis of VMI disorders.
5. Outline the characteristic appearance of full-thickness macular hole (FTMH) versus lamellar macular hole (LMH).
6. Contrast pathogenesis, clinical characteristics, and SD-OCT appearance of tractional versus degenerative LMH.
7. Understand the differences in appearance, development, and prognosis in epiretinal membrane (ERM) versus lamellar hole-associated proliferation (LHEP).

Case Report

- 69-year-old white male with complaints of blurry vision/disturbances OD
  - Gradually worsening x 3-4 months
  - Objects appear to “jump down and to the right, then disappear”

- Ocular history
  - Longstanding macular hole OD, VMT OS x 10 years
  - Co-managed with retina specialist, last visit 2 weeks prior
  - Surgery not recommended

- Medical history
  - Hypertension, depression/anxiety
  - Amlodipine, buspirone (anxiolytic), Celexa, Effexor

Preliminary Testing

<table>
<thead>
<tr>
<th></th>
<th>OD</th>
<th>OS</th>
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<tbody>
<tr>
<td>Entering VA</td>
<td>20/80-2 (cc)</td>
<td>20/20-2 (cc)</td>
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<tr>
<td>Amsler Grid</td>
<td>Central scotoma</td>
<td>Central metamorphopsia</td>
</tr>
<tr>
<td>Pupils</td>
<td>Round, reactive, no APD</td>
<td>Round, reactive, no APD</td>
</tr>
<tr>
<td>CVF</td>
<td>FTFC</td>
<td>FTFC</td>
</tr>
<tr>
<td>EOMs</td>
<td>FROM</td>
<td>FROM</td>
</tr>
<tr>
<td>Cover Test</td>
<td>Orthophoria (distance) 2 XP’ (near)</td>
<td></td>
</tr>
</tbody>
</table>

Anterior Segment

<table>
<thead>
<tr>
<th></th>
<th>OD</th>
<th>OS</th>
</tr>
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<tbody>
<tr>
<td>Adnexa</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Clear</td>
<td>Clear</td>
</tr>
<tr>
<td>Conjunctiva</td>
<td>Pinguecula nasal/temp</td>
<td>Pinguecula nasal/temp</td>
</tr>
<tr>
<td>Cornea</td>
<td>Clear</td>
<td>3+ edema</td>
</tr>
<tr>
<td>Anterior Chamber</td>
<td>Deep &amp; quiet</td>
<td>Deep &amp; quiet</td>
</tr>
<tr>
<td>Iris</td>
<td>Flat &amp; intact</td>
<td>Flat &amp; intact</td>
</tr>
<tr>
<td>Lens</td>
<td>1+ Nuclear sclerosis</td>
<td>1+ Nuclear sclerosis</td>
</tr>
</tbody>
</table>

Fundus Photography
Review of Vitreous Physiology

**Type II collagen**
- Strength & impact resistance
- Densely packed at vitreous base

**Hyaluronic acid**
- "Scaffolding" - separates & suspends collagen
- Highest concentration in PVC
- Synthesized by hyalocytes

**Collagen-hyaluronic acid complex**
- Balances gel vs. water content
- Optical clarity, strength + elasticity

The Vitreoretinal Interface

- Vitreous cortex – ILM junction
- ILM = retinal basal membrane = Müller cell footplates
- Adhesion of collagen to ILM mediated by fibronectin + laminin
  - Strength of adhesion increases from posterior pole to vitreous base

Vitreous Aging

- Disruption of collagen-hyaluronic acid complex
- Liquification of gel vitreous

- Increased cross-linking between collagen fibrils
- Collagen clumping with adjacent lacunae

- Vitreoretinal adhesions weaken
- Separation of vitreous cortex from ILM
- Vitreous collapses anteriorly

Sites of Vitreoretinal Adhesion

1. Vitreous base (strongest)
2. Posterior lens capsule
3. Optic disc
4. Macula
5. Blood vessels (weakest)

Vitreofoveal detachment with vitreopapillary adhesion

Stage 1: Perimacular dehiscence

Stage 2: Perifoveal dehiscence

Stage 3: Complete detachment
Anomalous Posterior Vitreous Detachment

- Disruption in normal vitreous aging
- Liquefaction outpaces normal weakening of VRA
- Anterior migration of vitreous body despite persistent retinal adhesion

Vitreomacular Adhesion (VMA)
Incomplete PVD + normal fovea

Vitreomacular Traction (VMT)
Incomplete PVD + disruption of fovea

Vitreouschisis w/ partial thickness PVC adherent

Axial Traction
Premacular Membrane
Tangential Traction

Typical Epiretinal Membrane
Fibrocellular proliferation of thick, non-contractile membrane on inner retina
- Histopathology: primarily gliotic tissue from Müller cells
- Microbreaks in ILM ➞ migration/proliferation of intraretinal glial cells
- Manifestation of chronic, severe gliosis
  - Increases local inflammation and neurodegeneration
  - May occur independently or concurrently with ERM

Lamellar Hole–Associated Epiretinal Proliferation (Atypical Epiretinal Tissue)
Fibrocellular proliferation of thin, highly contractile membrane on inner retina
- Histopathology: primarily myofibroblasts with contractile properties
- Tractional stress induces chronic inflammation & cytokine production
  - Fibroblast growth factor, nerve growth factor
  - Stimulate proliferation of residual PVC cells
Full-Thickness Macular Hole (FTMH)

- Full-thickness foveal break of the neurosensory retina extending from ILM to RPE

Lamellar Macular Hole

- Partial-thickness foveal break of the inner neurosensory retina not extending to the RPE

Tractional LMH

- Displaced retinal tissue
- Significant schisis between OPL and HFL
  - No disruption of Henle’s fibers
- Associated with tractional ERM

Degenerative LMH

- Loss of retinal tissue
- Intraretinal cavitations between IPL and HFL
  - Disruption of Henle’s fibers
- Slow, chronic retrograde degeneration of foveal PR, bipolar, & horizontal cells
  - Worse VA than tractional
- Associated with LHEP (In 98.5%)

<table>
<thead>
<tr>
<th>Tractional</th>
<th>Degenerative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thick, contractile ERM</td>
<td>Non-contractile epithelial prolifereation</td>
</tr>
<tr>
<td>Extrinsic to the fovea</td>
<td>Located at foveal edges</td>
</tr>
<tr>
<td>Sharp schisis between IML and OPL</td>
<td>Round-edged intraretinal cavitation</td>
</tr>
<tr>
<td>Inner/outer retinal diameter &lt;1.2</td>
<td>Inner/outer retinal diameter &gt;1.2</td>
</tr>
<tr>
<td>Intraretinal cystoid spaces</td>
<td>Foveal bump</td>
</tr>
<tr>
<td>Intact ellipsoid zone</td>
<td>Disruption of ellipsoid zone</td>
</tr>
</tbody>
</table>

Our Patient

- Thick, medium reflectivity
- Intraretinal cysts
- No PR disruption
- Foveal bump
- Round-edged cavitation

Optical coherence tomography angiographic findings of lamellar macular hole: comparisons between tractional and degenerative subtypes

- Superficial capillary plexus (SCP) runs through the NFL, GCL, and IPL
- Disruption of the VMI may affect superfiical retinal vasculature
- Mechanism of disruption may provide insight about pathophysics of LMH

FAZ area | Smaller | Larger |
Foveal VD | Higher | Lower |
Parafoveal VD | Lower | Lower |
VDI | Lower | Lower |

FAZ = foveal avascular zone, VD = vessel density, VDI = vessel density index

In tLMH, microvascular structure was restored 6 months after surgery
- NOT in dLMH

- Smaller foveal and parafoveal VD highly correlated with BCVA in dLMH
- Hypoperfusion to photoreceptors
Management Options

- Observation
- Jetrea (ocriplasmin)
- Pars Plana Vitrectomy

Management of Full-Thickness Macular Hole

<table>
<thead>
<tr>
<th>Gass Stage 1</th>
<th>Gass Stage 2</th>
<th>Gass Stage 3</th>
<th>Gass Stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression Rate</td>
<td>50%</td>
<td>35%</td>
<td>&lt;3%</td>
</tr>
<tr>
<td>Progression Rate</td>
<td>40% to FTMH</td>
<td>75%</td>
<td>50%</td>
</tr>
<tr>
<td>PPV</td>
<td>Commonly monitored</td>
<td>~90% success</td>
<td>~90%</td>
</tr>
<tr>
<td></td>
<td>ILM peel necessary</td>
<td>90-95%</td>
<td></td>
</tr>
</tbody>
</table>

Macular Hole Indices

- Macular hole index (MHI): \( \frac{BD}{MHH} \) Considers axial + tangential traction
- Hole forming factor (HFF): \( \frac{BD}{MHH} + \text{MD} \) Considers axial + tangential traction
- Diameter hole index (DHI): \( \frac{MD}{BD} \) Considers tangential traction only
- Max traction when min diameter = base diameter
- Tractional hole index (THI): \( \frac{MHH}{MD} \) Considers A-P > tangential traction

Strong correlation with 3-month post-op VA

Management of Lamellar Macular Hole

- Typically, anatomically and functionally stable
  - 13-21% enlarge in diameter after 18-24 months
  - Monitored → rarely progress to FTMH
- tLMH: better post-surgical outcomes
  - Release of traction restores normal anatomy
  - No consensus on when to initiate treatment
  - Worsening VA/metamorphopsia
  - Increased central thickness on OCT
  - Enlargement of hole

Potential Post-Surgical Outcomes

- Inadvertent ILM disruption during ILM peel → damage to Müller cells
  - May lead to FTMH development
- Possible factors increasing risk of surgical failure:
  - Location of ERM
  - Presence of LHEP

Pars Plana Vitrectomy with ILM Peel

- Mechanism
  - Release of traction
  - Stimulation of fibroglial proliferation
    - “Plugs” the hole
    - Hyperplastic RPE and fibroglial cells in spontaneously closed MHs
- Indications
  - Symptomatic VMT
  - Tractional LMH
  - Stage 2 or greater FTMH
  - Highly variable treatment outcomes

Central scotoma
Worse preoperative visual acuity
Larger hole diameter
Thinner retina
Higher incidence of ellipsoid disruption

LHEP is associated with...

LHEP influence on rate of macular hole closure remains controversial

EN-FACE IMAGING OF ATYPICAL EPIRETINAL TISSUE IN LAMELLAR MACULAR HOLE

Factors associated with ΔBCVA:
- Maximum diameter of LMH
- Area of LHEP on en-face OCT
  - Correlates with extent of foveal cavitation
  - <1.12 mm² associated with improved BCVA

ASSOCIATION BETWEEN EPIRETINAL MEMBRANE, EPIRETINAL PROLIFERATION, AND PROGNOSIS OF FULL-THICKNESS MACULAR HOLE CLOSURE

HME-ERM + EP
- Risk of unfavorable closure & surgical failure
- Mechanical damage & chronic inflammation of cells participating in hole healing

References


