Macular Thickness Changes in Post-Acute Branch Artery Occlusion Using Optical Coherence Tomography

Abstract: A branch retinal artery occlusion can often mimic glaucomatous changes in peripapillary RNFL scans and visual field testing. Utilization of macular thickness scans can help differentiate a post-acute retinal arterial obstruction from true glaucomatous atrophy.

I. Case History
   A. Demographics: 71 year-old Caucasian female
   B. CC: Decreased vision with a “cloud hanging over the top half” of her vision in the right eye that presented upon awakening.
   C. Ocular History:
      • Ocular Hypertension
      • Anatomically narrow angles s/p LPI OU
      • Diabetes Mellitus, Type II without retinopathy OU
      • Dry Eye Syndrome
   D. Medical History:
      • Diabetes Mellitus, Type II
      • Hypertension
      • Hyperlipidemia
      • Deep Vein Thrombosis
      • Asthma
      • Sleep Apnea
      • Hypothyroidism
   E. Medications:
      • Glipizide 10 mg
      • NovoLog 10 units
      • Hydrochlorothiazide (HCTZ) 25 mg
      • Aspirin 325 mg (started after cardiac workup)
      • Simvastatin 40 mg
      • Montelukast 10 mg
      • Albuterol PRN
      • Levothyroxine 0.1 mg

II. Pertinent findings
   A. Clinical
      • BCVA: 20/20-2 OD, 20/30+2 OS NIPH
      • CVF: Superior-temporal restriction OD, normal OS
      • GAT-IOP: 20 OD, 20 OS
      • SLE: Patent LPI @2:00 OD and @11:00 OS; 1+NS OU
      • DFE:
         • OU: Weiss ring
• OD:
  • 0.65 x 0.60 C/D in 2.2mm VDD, thin superior rim, vessel baring inferiorly
  • Choroidal nevus, temporal to FAZ
  • Hollenhorst plaque, inferior-temporal arcade near ONH
• OS:
  • 0.70 x 0.65 C/D in 2.2 mm VDD; thin superior rim
  • Choroidal nevus, along inferior-temporal arcade
• Pertinent negatives:
  • (-)CSME
  • (-)Neovascularization
  • (-)Hemes/CWS/Exudates

B. Physical
• Alert and oriented
• Mood/Affect appropriate

C. Laboratory studies
• Unremarkable: CBC, CMP, Lipid Profile, Thyroid Panel
• HbA1c: 8.5%; Glucose: 169

D. Radiology studies
• Carotid Doppler: 70% carotid stenosis bilaterally
• MRA (neck): Bilateral disease at carotid bulbus w/out significant ICA stenosis

E. Others
• Fluorescein angiogram: Hollenhorst plaque along inferior trunk of retinal artery (OD); poor/limited perfusion distal to plaque; staining of blood vessel.
• OCT RNFL (2 month f/u):
  • OD: Global thickness of 84um with inferior-temporal thinning
  • OS: Global thickness of 90um with no flag sectors
• OCT Posterior Pole Analysis (2 month f/u):
  • OD: Marked retinal thinning inferiorly with loss of inner retinal layer stratification. Significant intra-eye asymmetry of 53um (inferior<superior)
  • OS: unremarkable
• HVF 24-2 (2 month f/u):
  • OD: NEW Dense superior defect (PSD: 12.36)
  • OS: unremarkable
• HVF 10-2 (2 month f/u):
  • OD: Dense superior defect involving central fixation (PSD: 13.82)
  • OS: unremarkable
III. Differential Diagnosis
A. Primary: Branch retinal artery occlusion, OD
B. Others: CRAO, cilioretinal artery obstruction

IV. Diagnosis & Discussion
A. About the condition
   • Branch retinal artery occlusions (BRAO) represent approximately 38% of all acute retinal artery obstructions.\(^1\) The majority of branch retinal artery occlusions are caused by an embolus, which can be of cholesterol (Hollenhorst), calcific, or platlet-fibrin origin.\(^2\) The most common type of embolus is a Hollenhorst plaque, however potential embolic sources are found in less that 40% of cases.\(^3\) Temporal arterial circulation is more commonly affected.\(^4\) The mean age of onset is 60 years of age, and approximately 50-60% of patients have concurrent systemic arterial hypertension.
   • The presentation is with sudden and profound painless altitudinal or sectoral visual field loss. However, it can go unnoticed, particularly if central vision is spared.\(^5\) A BRAO that involves the central macular region will cause significant decrease in vision and often a relative afferent pupillary defect. Visual prognosis after BRAO appears to be correlated to presenting visual acuity, with most patients obtaining little to no improvement over time.\(^6\)
   • Examination of the retina in the acute phase will reveal arteriolar attenuation, retinal whitening secondary to edema, and often the presence of one or more emboli that are usually located at a bifurcation point. While a diagnosis can be made without ancillary testing, optical coherence tomography (OCT) will initially reveal thickening and hyper-reflectivity of the inner retinal layers.\(^7\) Intravenous fluorescein angiography (IVFA) will show delayed arterial filling and hypofluorescence in the area of retinal edema.\(^8\)

B. Unique Considerations
   • In the chronic stages of a BRAO, recanalization of the obstructed arterial vessel can occur, leaving subtle to no ophthalmoscopic signs of the occlusive event. However, signs via OCT include atrophy of the corresponding inner retina, as well as the peripapillary retinal nerve fiber layer (RNFL).\(^9\)
   • Because patients without central macular involvement can often go undiagnosed in the acute phase, the possibility of subtle ophthalmoscopy signs, and sectoral peripapillary RNFL and inner retinal atrophy more likely in one of the temporal quadrants – a BRAO can produce glaucoma-like changes in ancillary testing such as visual field testing and
OCT RNFL measurements. This can lead to confounding results in patients where these tests are used to diagnose or manage glaucoma patients.

- However, utilization of an OCT macular thickness scan can assist in differentiating the two diseases. Three main macular OCT features that distinguish a post-acute BRAO from true glaucomatous atrophy include:
  - Complete inner retinal atrophy with loss of normal stratification of the inner retinal layers
  - Loss of normal foveal depression
  - Marked thinning of the involved retina

V. Treatment & Management
   A. Treatment Options
      - There is no proven treatment for a branch retinal artery occlusion. Retinal neovascularization is very rare, and iris neovascularization does not occur. However, a medical work-up is indicated (see below).
   B. Patient Management (Work-up):
      - Within eye exam:
         - Blood pressure
         - Pulse
         - Carotid bruit auscultation
      - Imaging:
         - Carotid Doppler
         - Echocardiography
         - Consider IVFA
      - Initial Labs:
         - CBC
         - ESR
         - Pt/Ptt
         - Lipid Profile
         - RPR/FTA-ABS
         - Homocysteine
         - SPEP
      - Full cardiac work-up through PCP

VI. Conclusion
   A. Our patient, who is also a glaucoma suspect, is an ideal case to illustrate the marked macular thickness differences in these two diseases.
      - Acute BRAO stage: A macular raster will show an edematous inner retina with an associated hyper-reflectivity of the inner layers.
• **Post-acute (chronic) BRAO stage:** Long-term follow-up with a macular raster will show marked thinning of entire retinal segment, along with complete loss of normal stratification of the inner retinal layers, and changes to normal foveal depression.