Grand Rounds 07
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Moderator: Gerald Selvin, O.D.

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Disclosure Statement:
Speakers include disclosures in each presentation. I have none.
Non-Ischemic Central Retinal Vein Occlusion With Associated Paracentral Acute Middle Maculopathy

Mayur Bhavsar OD, FAAO
VA NJ Healthcare System
Disclosure Statement:
Nothing to disclose
Case History

• 54 year old Caucasian female presents with a sudden patchy loss of vision in her right eye upon waking. She indicates mild inferior nasal blur.

• The patient was stressed due to her mother being in the hospital.

• Medical history is unremarkable. Reports high blood pressure readings in the past.

• No medications but taking a daily multivitamin, 1000 mcg of Vitamin B12, 2000 units of Vitamin D3, 5000 mcg of Biotin, and 5 mg of Melatonin.
Exam findings

- VA with current glasses:
  - OD: -2.75 -0.25x 180 20/20
  - OS: -2.75 sph 20/20
- Pupils: OD: 5 mm, OS 5 mm (-) APD
- Confrontation VF: Normal OD/OS
- EOM- Full and smooth
- Slit Lamp Examination- Unremarkable
  - Trace Nuclear Sclerotic Cataracts OU
- Tonometry: OD: 21 mm Hg, OS: 20 mm Hg
Fundus findings

OD: 0.45 round and healthy optic nerve.
Macula flat and healthy
Large blot hemorrhage superior temporal to the macula with a multiple adjacent dot hemorrhages temporal. There are also scattered dot and blot hemorrhages along the superior temporal arcade and one blot hemorrhage along the inferior temporal arcade.
The vessels are dilated and tortuous. The periphery is normal.

OS: 0.35 round and healthy optic nerve.
Macula flat and healthy
The left posterior pole and blood vessels are unremarkable.
The periphery is normal.
Diagnosis and Plan

1) Branch retinal vein occlusion vs Hemiretinal Vein occlusion- Pt educated to see retinal specialist for IVFA and OCT. Edu on better control of blood pressure with Yoga and meditation

2) Suspected Hypertension with retinopathy OU- Pt educated on good blood pressure control with diet and exercise. Edu to have BP checked today and have a full physical with blood work performed by primary care physician.

3) Trace Cataracts- No tx indicated. Monitor

4) Presbyopia- Continue with present glasses
Blocking defects corresponding to intraretinal hemorrhages. No evidence of vascular leakage or non-perfusion noted. Arterial and venous phases not captured due to poor patient cooperation.
IVFA OS

5 mins, 57 secs--Unremarkable
OCT Macular Cube

OD: No macular edema. Central thickness is 261 microns

OS: No macular edema. Central thickness is 236 microns
OCT Raster Line
Raster line OD
Diagnosis and Plan

• Combined Non-Ischemic CRVO with Parcentral Acute Middle Maculopathy
  • Recommend systemic workup: Carotid ultrasound, Cardiac Echo, and Lipid profile.
  • Discussed BP control
• Follow up 1 month
Follow up-6 weeks after initial presentation

- Pt reports that vision has normalized subjectively
- Pt reports having extensive evaluation of embolic sources– All normal.
- VA stable at 20/20 OD, OS, OU
- Pupils: OD: 5 mm, OS 5 mm (-) APD
- Confrontation VF: Normal OD/OS
- EOM- Full and smooth
- Slit Lamp Examination- Unremarkable
  - Trace Nuclear Sclerotic Cataracts OU
- Tonometry: OD: 18 mm Hg, OS: 19 mm Hg
Posterior Pole

- C/D- OD: 0.55, OS: 0.4– No disc edema or pallor. No NVD OU
- Vitreous- Clear OU
- Retinal vessels- Normal caliber OU
- Periphery: No peripheral pathology. No holes/tears.
**OCT Macular Cube Visit 2**

- **OD**: No macular edema. Central thickness is 237 microns
- **OS**: No macular edema. Central thickness is 246 microns
Change in OCT

- Visit 1 OD
- Visit 2 OD
Diagnosis and Plan

- Resolving Non-ischemic CRVO and PAMM- No treatment indicated. Re-educated on Blood Pressure control.
- Mild cataracts- Monitor
- Follow up 4 months
**Paracentral Acute Middle Maculopathy**

- Refers to band-like hyperreflective spectral-domain optical coherence tomography lesions mostly confined to the middle retina (at the junction of the OPL and INL and extending into the INL).
- Deeper form of the well-known cotton-wool spot.
- Not a disease entity but rather a Clinical Finding.
- Initially described as a variant of Acute Macular Neuroretinopathy (AMN).
Common PAMM associated conditions

• Non-Proliferative Diabetic Retinopathy
• Retinal Vein Occlusions (CRVO and HRVO)
• Retinal Artery Occlusions (BRAO, CRAO, Cilioretinal artery occlusion)
• Sickle Cell Retinopathy
• Purtscher Retinopathy
• Rare autoimmune conditions
• Can theoretically be seen in any patient with retinal vascular disease or systemic vasculopathic risk factors.
Symptoms and Characteristic Ocular Findings

- Symptoms— Often include Paracentral Scotoma OR non-specific visual disturbance
- Vision varies between 20/20 to Hand Motion. Depends on the underlying retinal diagnosis.
- Fundus findings— NONE or ill-defined whitish retinal lesion
- IVFA- Typically NONE
- Near-Infrared Reflectance- Dark Grey wedge-like or petaloid lesions
- Spectral Domain OCT- Hyperreflective band at the junction of the OPL and INL and extending into the INL
PAMM causes

• Ischemia of the intermediate and deep capillary plexuses
• Watershed-like region—therefore greatest risk for ischemic insult
  • Oxygen demand of the macula, especially at the level of the INL, OPL and Photoreceptor inner segments, is higher than any other region in the retina
  • Parafoveally the retinal thickness is greatest, therefore oxygen diffusion from the choroid is more limited
en face OCT

• High-density raster pattern of rapidly acquired line scans which creates a 3-dimensional reconstruction of the macula.
• Provides segmented visualization of the various retinal and/or choroidal layer
• Can assist in visualizing patterns of ischemia in the intermediate and deeper retinal layers.
• PAMM presented with 3 different patterns of well-demarcated hyperreflectivity (Sridhar)
  • Arteriolar
  • Globular
  • Fern-like
Optical Coherence Tomography Angiography (OCTA)

- Novel and noninvasive technique for demonstrating the microvascular blood flow within the retina
- Produces depth-resolved evaluation of the reflectance data from retinal tissue
- PAMM shows the most changes in the Deep Capillary Plexus with some changes in the Superficial Capillary Plexus

A. Superficial Capillary Plexus, B. Deep Capillary Plexus, C. Choriocapillaris
Optical coherence tomography angiography and en face optical coherence tomography features of paracentral acute middle maculopathy. Inferior field cut in the setting of central retinal vein occlusion. Spectral-domain optical coherence tomography (OCT) demonstrated superior graying on near-infrared reflectance (Top left) with middle retinal hyperreflectivity in the corresponding area (Top right). Fluorescein angiography (Bottom left) showed areas of blockage reflecting preretinal and intraretinal heme and intact perfusion to superior macula. En face OCT (Bottom right, top row) at the level of the superficial (left), middle (middle), and outer (right) retina demonstrated band-like hyperreflectivity at the level of the middle retina. OCT angiography at the same levels (Bottom right, bottom row) is notable for absence of deep capillary plexus dropout at the level of the middle retina.
en face OCT of CRVO showing Fern-like ischemia (Ghasemi)

FIGURE 1. Color fundus photography (Top left) and fluorescein angiography (Top right) of a 36-year-old male patient illustrate findings consistent with the diagnosis of central retinal vein occlusion. Cross-sectional optical coherence tomography (OCT) (Middle) demonstrates characteristic paracentral acute middle maculopathy (PAMM). En face OCT (Bottom) segmented at the level of the inner nuclear layer and with vascular mapping illustrates a remarkable and precise perivenular distribution of fern-like PAMM with periarterial sparing.
Treatment

• NONE

• Patients that received intravitreal Anti-VegF for treatment of macular edema did not appear to impact the timeline to resolution of the PAMM. (Rahimy)

• We do not possess the tools for restorative intervention at the capillary level, and that’s why there is no specific treatment for PAMM of AMN (Dansingani)
Take Home PAMM

• Refers to band-like hyperreflective spectral-domain optical coherence tomography lesions mostly confined to the middle retina.
• It is caused by ischemia of the intermediate and deep capillary plexuses.
• With the advent of imaging techniques such as en face OCT and OCTA we can visualize the level and degree of ischemia.
• If you have a patient with vague central vision symptoms, you should perform an SDOCT and look at the INL and ONL layers closely.
Works Cited


https://doi.org/10.3928/23258160-20131101-06


https://entokey.com/normal-retinal-anatomy-and-basic-pathologic-appearances

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Case of Retinal Emboli – Ocular & Cardiovascular Associations; and the “Controversy” of Carotid Imaging for Asymptomatic Retinal Emboli

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Optometrist
VA Palo Alto Health Care System
Disclosures: None
Objectives

• Review case of retinal emboli and its complications
• Review retinal emboli types and ocular & systemic associations
• Review current literature and guidelines for management
Case: 69M

• POV: Routine eye exam
• CC: none - stable vision and no complaints

• Ocular History:
  • Mild cataracts OU
  • Low risk glaucoma suspect due to optic nerve head appearance OU
  • Epiretinal membrane OD, not visually significant

• PMHx:
  • Impaired fasting glucose, dyslipidemia, rhinitis
Examination Findings

• BCVA 20/20 OU
• PERRL, no RAPD
• Confrontations unremarkable
• SLE remarkable for mild nuclear sclerotic cataracts OU
• IOP 18/19 mmHg at 1018
Differential Diagnoses:

• Retinal emboli
  • Hollenhorst
  • Platelet-fibrin
  • Calcific plaque

• Other:
  • Talc Retinopathy
  • Tumor, fat, septic
Retinal Emboli

• 1.4% of adults >40yo (Blue Mountain Eye Study, Beaver Dam Eye Study, LALES, SEED)
  ▪ 20-30% of eyes have numerous emboli
  ▪ Transient; 90% resolve (Beaver Dam Eye Study)

• May also find:
  ▪ Transient monocular visual loss/AF/TIA
  ▪ Central or branch retinal artery occlusions
  ▪ Cotton wool spots
  ▪ Localized areas of periarteriolar sheathing

• 75% are asymptomatic
Pathogenesis

• Dislodge from atheroma of
  • Carotid artery (60-70%)
  • Heart valves
  • Ascending aorta
• Vessel obstruction distal to atheroma

Spalton et al. Elsevier. 2005
Hayreh: Retinal Artery Occlusion and Systemic Disease

- Carotid artery disease - most common cause of RAO (embolism)
  - Most commonly due to presence of plaques and less frequently stenosis
- Heart
  - Aortic and mitral valvular lesions, patent foramen ovale, tumor of left atrium and myxoma

<table>
<thead>
<tr>
<th></th>
<th>ICA Stenosis &gt;50%</th>
<th>ICA Plaque Presence</th>
<th>Abnormal Echocardiogram</th>
<th>Embolic Source on Echocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>BRAO</td>
<td>30%</td>
<td>66%</td>
<td>58%</td>
<td>42% embolic</td>
</tr>
<tr>
<td>CRAO</td>
<td>34%</td>
<td>71%</td>
<td>70%</td>
<td>52% embolic</td>
</tr>
</tbody>
</table>

- High prevalence of cardiovascular disease in CRAO as well as BRAO
- Emboli may come from either carotid artery or heart or both

Risk Factors and Associations of Retinal Emboli

- Age
- Hypertension
- Smoking
- Plaques and carotid stenosis
- Chronic Kidney Disease
- Cardiovascular Disease
  - Stroke
  - Ischemic Heart Disease
  - Hypercholesterolemia
  - Diabetes

<table>
<thead>
<tr>
<th>Age Group (years)</th>
<th>Los Angeles Latino Eye Study</th>
<th>Blue Mountains Eye Study*</th>
<th>Age Group (years)</th>
<th>Los Angeles Latino Eye Study</th>
<th>Beaver Dam Eye Study*</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60</td>
<td>0.3%</td>
<td>0.8%</td>
<td>40 to 49</td>
<td>0.3%</td>
<td>0.1%†</td>
</tr>
<tr>
<td>60 to 69</td>
<td>0.5%</td>
<td>1.4%</td>
<td>50 to 59</td>
<td>0.3%</td>
<td>0.7%</td>
</tr>
<tr>
<td>70 to 79</td>
<td>1.1%</td>
<td>2.1%</td>
<td>60 to 69</td>
<td>0.5%</td>
<td>1.6%</td>
</tr>
<tr>
<td>≥ 80</td>
<td>0%</td>
<td>1.5%</td>
<td>≥ 70</td>
<td>0.9%</td>
<td>2.0%</td>
</tr>
</tbody>
</table>

*The Blue Mountains Eye Study data were obtained from Mitchell and associates and the Beaver Dam Eye Study data were obtained from Ronald Klein (personal communication, 2007).
†Age range, 43 to 49 years.
• Stroke – major cause of death and disability
  • Large burden on resources
  • 2\textsuperscript{nd} leading cause of death in world
• N = 4926
• 1.3\% prevalence of retinal arteriolar emboli
• Persons with retinal emboli at baseline had an increased risk of dying with stroke vs. without emboli

\[
\text{Table 4. Relation of Retinal Arteriolar Emboli at Baseline to Stroke and Ischemic Heart Disease Deaths in the Beaver Dam Eye Study}\]

<table>
<thead>
<tr>
<th>Retinal Arteriolar Emboli*</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>4795</td>
<td>61</td>
</tr>
<tr>
<td>Person-years</td>
<td>34 010.2</td>
<td>377.6</td>
</tr>
<tr>
<td>Stroke deaths</td>
<td>91</td>
<td>6</td>
</tr>
<tr>
<td>Stroke deaths per 1000 person-years</td>
<td>2.7</td>
<td>15.9</td>
</tr>
<tr>
<td>Ischemic heart disease deaths</td>
<td>232</td>
<td>5</td>
</tr>
<tr>
<td>Ischemic heart disease deaths per 1000 person-years</td>
<td>6.8</td>
<td>13.2</td>
</tr>
</tbody>
</table>

*In 8 persons with stroke-related deaths and 10 persons with cardiovascular disease–related deaths, the retinal emboli status at baseline was not known.
8.5% annual stroke risk vs. 0.8% in controls (matched for age, hypertension, diabetes, and smoking)

10x higher stroke risk

Higher rate of carotid stenosis and ischemic heart disease
Management Options

• Carotid Studies
  • Carotid ultrasonography, MRI, CT, carotid auscultation

• Cardiac Studies
  • Echocardiogram (electrocardiogram, Holter monitoring)

• Interventions?
  • Medical: triple medical therapy
    • Statin, antiplatelet therapy and blood pressure-lowering therapy
  • Surgical
    • Carotid Endarterectomy (CEA), stenting, valve replacement
Carotid Intervention Clinical Trials

• Stenting vs. CEA for Asymptomatic Carotid Stenosis:
  • Ipsilateral Stroke
    • 2.2% after stenting vs. 2.7% after CEA - ACT-1
    • 6.9% after stenting vs. 5.6% after CEA – CREST

• The Asymptomatic Carotid Atherosclerosis Study (ASCT)
  • 1662 patients with asymptomatic carotid artery stenosis of >60%
  • Randomized to conventional medical management or surgery
  • The estimated 5-year ipsilateral stroke rate:
    • 11% medical treatment group
    • 5.1% CEA group
  • 2.7% 30-day morbidity and mortality including complications of angiography
### USE OF CAROTID ENDARTERECTOMY IN SYMPTOMATIC PATIENTS

<table>
<thead>
<tr>
<th>Stenosis (%) ICA angiographic</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>70-99%</td>
<td>• CE is established as effective for recently symptomatic (within previous 6 months) patients with 70-99% ICA angiographic stenosis (Level A).</td>
</tr>
</tbody>
</table>
| 50-69%                       | • CE may be considered for patients with 50-69% symptomatic stenosis (Level B) but the clinician should consider additional clinical and angiographic variables (Level C). See tables below.  
• It is recommended that the patient have at least a five year life expectancy and that the perioperative stroke/death rate should be <6% for symptomatic patients (Level A). |
| <50%                         | • CE should not be considered for symptomatic patients with <50% stenosis (Level A).  
• Medical management is preferred to CE for symptomatic patients with <50% stenosis (Level A). |

### USE OF CAROTID ENDARTERECTOMY IN ASYMPTOMATIC PATIENTS

<table>
<thead>
<tr>
<th>Stenosis (%) ICA angiographic</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-99%</td>
<td>• It is reasonable to consider CE for patients between the ages of 40 and 75 years and with asymptomatic stenosis of 60-99% if the patient has an expected five year life expectancy and if the surgical stroke or death frequency can be reliably documented to be &lt;3% (Level A). The five year life expectancy is important since perioperative strokes pose an up front risk to the patient and the benefit from CE emerges only after a number of years.</td>
</tr>
</tbody>
</table>
The Reasons for Concern

• Expensive equipment that requires time for training and interpretation
• Unnecessary investigations
• Risk of investigations
• Unnecessary interventions
  • Perioperative stroke risk
• Cognitive decline
Symptomatic vs. Asymptomatic

- Do they represent different disease processes?
  - Different mechanism
  - Smaller emboli to impair vision than other brain functions

- What is the positive predictive value for testing?

- Sharma et al – Echocardiographic Studies for Acute Retinal Artery Occlusions
Less Common Sources of Emboli: Heart + Aorta

• Long-term all-cause mortality: 12% w/emboli vs. 4% without emboli
  • Associated cardiovascular deaths not significantly observed
  • No relationship to incidence of fatal myocardial infarction - BDES

• The source of embolism in amaurosis fugax and retinal artery occlusion – Mouradian et al
  • 11 of 41 patients had stenosis of ICA (27%)
  • 1 of 41 patients had embolism of cardiac source (2%)
  • 2/3 without known cause

• Aortic Arch Disease
  • Transesophageal echocardiogram for symptomatic embolism or younger presentation

Mouradian et al. Journal of Neuroimaging. 2002
Case - Study Results:

Carotid Ultrasound Impression:

1. Normal left carotid artery with less than 50% stenosis and patent vertebral artery w/antegrade flow.
2. Right internal carotid artery with high grade stenosis of greater than 80%. Patent vertebral artery with antegrade flow. Will need vascular surgery evaluation.

Cardiac studies normal
Vascular Surgery Consult:

• “Because the patient has a high risk lesion, we recommend a right carotid stent.”

• Statin therapy and daily ASA 81mg

• Uncomplicated cannulation of right ICA with emboshield protection device, 6mm stent placement, and post stent angioplasty with 5x20mm balloon

• Enrollment into “Long-term Neurocognitive Sequelae of Subclinical Microembolization During Carotid Interventions”
Current Trends

• Improved medical interventions: “best medical therapy” BMT
• Decreased procedural risks  
  • Improvement in devices, technical experience & medical therapy
• Transesophageal echocardiogram vs. transthoracic echocardiogram
• Future Studies  
  • Asymptomatic Carotid Trial-2 (ASCT-2) in 2020  
  • Trial recruitment in Asia and Australia
Conclusions:

• Management include non-surgical and surgical interventions

• **DO:**
  • Carotid work-up if symptoms are present
    • Additional medical consultation if calcific or platelet emboli present
  • Stratify stroke risk and educate patient
    • Smoking, cholesterol
  • Educate about stroke symptoms including AF/TIA. If symptoms present:
    • Systemic work-up including carotid and cardiac studies
    • Ocular examination

• **CONSIDER:**
  • Carotid studies in asymptomatic individuals to further stratify stroke risk
  • Cardiac studies especially in younger patients though less common
  • Vascular surgery consultation
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Arruga J, Sanders MD Ophthalmologic findings in 70 patients with evidence of retinal embolism. Ophthalmology 1982;89:1336-1347


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Treatment of Pediatric Lyme Meningitis: Differing Approaches

MICHELLE L. WERTELET, OD, FAAO, DIPLOMATE ABO
Disclosure Statement:
Nothing to disclose
Reported Cases of Lyme Disease—United States, 2016

Each dot represents one case of Lyme disease and is placed randomly in the patient’s county of residence. The presence of a dot in a state does not necessarily mean that Lyme disease was acquired in that state. People travel between states, and the place of residence is sometimes different from the place where the patient became infected.
Case One

Patient demographics
- 5-year-old female

Chief complaint
- Referred by pediatrician due to positive Lyme results

History of present illness
- Severe headaches for the last few weeks
- Fatigue
- Decreased appetite

Medications
- None

One week ago visited Children’s Hospital emergency department
- Headache for 2 weeks
- Fever of 104°
- Intermittent rash
- No known tick exposure
Case One

VA
- OD 20/20, OS 20/20

Sensorimotor
- Ocular motilities full OU
- Orthophoria at distance and near

Slit lamp examination
- Normal OU

Dilated fundus examination
- 2+ Disc edema OU

Diagnosis
- High suspicion of Lyme meningitis
- Papilledema OU

Plan
- Consulted with pediatrician
- Patient to go to Children’s Hospital ED
- Neurology consultation
- Probable MRI
- Probably lumbar puncture
Case One

Diagnostic Testing
- MRI
  - No mass lesion
- Lumbar puncture
  - Mild lymphocytic pleocytosis
  - Opening pressure of 26 cm H₂O

Treatment
- IV ceftriaxone
  - 2 doses while inpatient
- Doxycycline
  - 32 mg PO BID x 28 days
- Acetazolamide
  - 120 mg PO BID x 2 months
- For moderate pain, use acetaminophen
- For severe pain, use ibuprofen
Case One

One week later

Medications
- Doxycycline BID
- Acetazolamide BID

VA
- OD 20/20, OS 20/20

Sensorimotor
- Ocular motilities full OU
- Orthophoria at distance and near

Slit lamp examination
- Normal OU

Dilated fundus examination
- Trace disc edema OU

Two months later

Medications
- Acetazolamide qhs, with 2 doses left

VA
- OD 20/20, OS 20/20

Sensorimotor
- Ocular motilities full OU
- Orthophoria at distance and near

Slit lamp examination
- Normal OU

Dilated fundus examination
- ON flat, pink, distinct OU
Case Two

Patient demographics
- 13-year-old male

Chief complaint
- Double vision and headaches

History of present illness
- Ill for the last 6 weeks
- Severe headache
- Low grade fever
- Whole body aches

- Eight days ago, seen in Children’s ED
- CT of head
  - No mass effect
- Lab work
  - Normal, but awaiting Lyme results
- Two days ago, Lyme positive
- Started doxycycline 75 mg BID x 28 days
Case Two

VA
- OD 20/20, OS 20/25

Sensorimotor
- Ocular motilities full OU

Slit lamp examination
- Normal OU

Dilated fundus examination
- Normal, No papilledema

Diagnosis
- Esotropia with diplopia
- Suspected mass lesion
- Lyme disease

Plan
- Consulted with pediatrician
- Recommended MRI of brain and orbits
Case Two

MRI of brain and orbit
- No hemorrhage or mass lesion
- Significant artifact from orthodontics
Case Two

Chief complaint
- Horizontal, constant diplopia
- Closes OD to see
- Headaches

Medications
- Doxycycline BID

VA
- OD 20/20, OS 20/20

Sensorimotor
- Ocular motilities

Dilated fundus examination
- Temporal ON elevation with blurred margins OU
Case Two

Diagnosis
◦ Right CN VI palsy with early papilledema in the setting of Lyme disease

Plan
◦ Consulted with Children’s Hospital infectious disease
◦ Suspected early disseminated Lyme meningitis
◦ Discontinue doxycycline
◦ Start amoxicillin 500 mg PO TID x 28 days
◦ Follow up with infectious disease in 2 weeks
◦ Any worsening of any symptoms, go straight to Children’s emergency department
## Case Two

<table>
<thead>
<tr>
<th>Five weeks later</th>
<th>Three months later</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VA</strong></td>
<td><strong>VA</strong></td>
</tr>
<tr>
<td>◦ OD 20/20, OS 20/20</td>
<td>◦ OD 20/20, OS 20/20</td>
</tr>
<tr>
<td><strong>Sensorimotor</strong></td>
<td></td>
</tr>
<tr>
<td>◦ Ocular motilities full OU</td>
<td>◦ Ocular motilities full OU</td>
</tr>
<tr>
<td>◦ Orthophoria at distance, E(T)’ 5</td>
<td>◦ Orthophoria at distance and near</td>
</tr>
<tr>
<td><strong>Slit lamp examination</strong></td>
<td><strong>Slit lamp examination</strong></td>
</tr>
<tr>
<td>◦ Normal OU</td>
<td>◦ Normal OU</td>
</tr>
<tr>
<td><strong>Dilated fundus examination</strong></td>
<td><strong>Dilated fundus examination</strong></td>
</tr>
<tr>
<td>◦ Normal OU</td>
<td>◦ Normal OU</td>
</tr>
</tbody>
</table>
Bonus Case
Bonus Case
Abnormal Findings on Diagnostic Tests

MRI of Brain and Orbits
- Nonspecific foci of T2 prolongation in the cerebral white matter
- Nerve root or meningeal enhancement

Lumbar puncture
- Lymphocytic pleocytosis
- Normal opening pressure in children < 28 cm H₂O
- Factors that may affect opening pressure
  - Depth of sedation
  - Sedation agent used
  - Leg position
Medications for Lyme Disease

- **Doxycycline**
  - Pediatric: 4 mg/kg BID x 10-21 days (Max 100 mg/dose) for early localized disease without neurological symptoms; treat x 28 days for Lyme arthritis (>8 years old and >45 kg)
  - Adult: 100mg PO BID x 10-21 days for early localized disease without neurological symptoms; treat x 28 days for Lyme arthritis

- **Amoxicillin**
  - Pediatric: 50 mg/kg/day PO TID x 14-21 days for early Lyme (Max 500 mg/dose)
  - Adult: 500 mg PO TID x 14-21 days for early Lyme

- **Ceftriaxone**
  - Pediatric: 50-75 mg/kg/day IV QD x 7-21 days (Max 2 g/24 hours)
  - Adult: 2 g IV QD
## Treatment Approaches

**CASE ONE**

- **Ceftriaxone IV**
  - 1830 mg, 45.75 ml, IV q24 hours

- **Doxycycline 25 mg/5 ml oral suspension**
  - 32 mg PO BID for 28 days

- **Acetazolamide 25 mg/ml oral suspension**
  - 120 mg PO BID for 2 months

**CASE TWO**

- **Doxycycline 25 mg/5 ml oral suspension**
  - 75 mg BID x 28 days

- **Amoxicillin 250 mg/5 ml oral suspension**
  - 500 mg PO TID x 28 days
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