Transient Monocular Blindness

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Transient Monocular Blindness

a Herald Symptom of Stroke to the Brain and / or Eye
Arterial supply of optic nerve and retina
Four types of monocular TMB

TMB Type I due to transient retinal ischemia
TMB Type II due to retinal vascular insufficiency
TMB Type III due to transient angiospasm
TMB Type IV - idiopathic
Identify Risk Factors

Cardiac / MI, Atrial Fib
Stroke – TIA
Hypertension
Hyperlipidemia
Diabetes Mellitus
Smoking
Obesity
Meticulous History of the Attack

- Onset
- Extent of vision loss
- Duration
- Speed of recovery
- Activity at the time
- Frequency of attacks
- Medications / time taken
# Types of TMB

<table>
<thead>
<tr>
<th></th>
<th>Type I</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
<td>Abrupt</td>
<td>Less rapid</td>
</tr>
<tr>
<td><strong>Visual loss</strong></td>
<td>All or partial</td>
<td>All or partial</td>
</tr>
<tr>
<td><strong>Length</strong></td>
<td>Seconds to minutes</td>
<td>Minutes to hours</td>
</tr>
<tr>
<td><strong>Recovery</strong></td>
<td>Complete</td>
<td>Complete</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
<td>Embolus to retinal vessel</td>
<td>Hypofusion due to carotid A. (int &amp; ext) occlusion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Type III</th>
<th>Type IV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
<td>Abrupt</td>
<td>Abrupt</td>
</tr>
<tr>
<td><strong>Visual loss</strong></td>
<td>Total or progressive</td>
<td>Partial</td>
</tr>
<tr>
<td><strong>Length</strong></td>
<td>Usually minutes</td>
<td>Seconds, minutes or hours</td>
</tr>
<tr>
<td><strong>Recovery</strong></td>
<td>Usually complete</td>
<td>Complete</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>Often</td>
<td>No</td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
<td>Angiospasm of ophthalmic</td>
<td>Unknown /Antiphospholipid antibodies</td>
</tr>
</tbody>
</table>
Ask about Associated Symptoms

- Headache / Neck pain
- Scalp tenderness
- Jaw claudication
- Fatigue
- Angina
- Palpitations
- Gait claudication
- Vertigo / Light headedness
List Medications

Anticoagulants (warfarin, heparin)
Antiplatelets (ASA, Plavix, ASA/Dipyridamal)
BP meds – time taken
Sildenafil (Viagra (EDD))
Cardiac meds
Statins (Lipitor etc)
Others
Viagra Associated Anterior Ischemic Optic Neuropathy

TMB Type I is one variety of an internal carotid artery distribution transient ischemic attack (TIA)
Pathology of surgically removed plaque in 23 cases of transient monocular blindness

<table>
<thead>
<tr>
<th>Diameter of residual lumen (mm)</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 or less</td>
<td>19 (90%)</td>
</tr>
<tr>
<td>1-2</td>
<td>0</td>
</tr>
<tr>
<td>2-3</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Mural thrombus</td>
<td>22 (96%)</td>
</tr>
<tr>
<td>Ulceration</td>
<td>13 (57%)</td>
</tr>
<tr>
<td>Internal carotid artery</td>
<td>11</td>
</tr>
<tr>
<td>Common carotid artery</td>
<td>3</td>
</tr>
<tr>
<td>Interplaque hemorrhage</td>
<td>15 (65%)</td>
</tr>
<tr>
<td>Cul-de-sacs</td>
<td>2</td>
</tr>
</tbody>
</table>

What to look for funduscopically after dilation of the pupil

Normal disc and fundus
Retinal emboli
BROA = visible embolus
Retinal infarct = cytoid body
Venous stasis retinopathy
Asymmetric hypertensive retinopathy
A low diastolic ophthalmic artery pressure
Ischemic disc swelling (AION)
## Sources of Emboli

<table>
<thead>
<tr>
<th>Source</th>
<th>Type</th>
<th>Patient Age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valves</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumatic disease</td>
<td>Platelet/calcium</td>
<td>Any age</td>
</tr>
<tr>
<td>Lupus</td>
<td>Platelet</td>
<td>Young woman</td>
</tr>
<tr>
<td>Acute or subacute endocarditis</td>
<td>Platelet</td>
<td>Damaged heart</td>
</tr>
<tr>
<td>Floppy mitral valve</td>
<td>Platelet</td>
<td>Any age; mostly women</td>
</tr>
<tr>
<td><strong>Chamber</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myxoma</td>
<td>Myxoma</td>
<td>Older adult</td>
</tr>
<tr>
<td>Mural thrombus</td>
<td>Platelet/clot</td>
<td></td>
</tr>
<tr>
<td><strong>Paradoxical Emboli</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patent Foramen Ovale</td>
<td>Platelet/clot</td>
<td>Any age</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>Platelet/clot</td>
<td>Any age</td>
</tr>
<tr>
<td><strong>Carotid Artery</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ulcerated plaque</td>
<td>Platelet/cholesterol ester</td>
<td>Older adult</td>
</tr>
<tr>
<td>Stenosis</td>
<td>Platelet</td>
<td>Any Age</td>
</tr>
<tr>
<td>Dissection</td>
<td>Platelet</td>
<td>Young woman</td>
</tr>
<tr>
<td>Fibromuscular dysplasia</td>
<td>Platelet</td>
<td></td>
</tr>
</tbody>
</table>
Internal Carotid Stenosis
Cardiac Sources

**Thromboembolism**

Myocardial Infarction - mural thrombus
Mitral stenosis ± atrial fibrillation
Vegetative valvular lesion, bacterial or non-bacterial
Mitral - annulus calcification
Prolapse of the mitral valve
Atrial myxoma
Patent foramen ovale
Atrial septal defect
Left atrial appendage thrombus

Transesophageal echocardiogram shows a large mobile thrombus in the left atrial appendage, extending into the body of the left atrium. Spontaneous echo contrast can be seen in the left atrium. (Courtesy of Thomas Binder, MD. University of Vienna)
Presence of aortic arch atherosclerotic lesions

- Embolic stroke patients: 42%
- Cryptogenic stroke patients: 61%
- Patients with neurologic disease: 5%
- Patients with cerebrovascular disease: 26%

Percentage of Patients
Presence of Patent Foramen Ovale

- **Patients > 55 years**
  - Cryptogenic strokes: 38%
  - Stroke with known causes: 8%

- **Patients < 55 years**
  - Cryptogenic strokes: 47%
  - Stroke with known causes: 4%

**Legend**
- Green: Cryptogenic strokes
- Pink: Stroke with known causes
TEE w/ contrast of a PFO

A. Diastole

B. Opacification of the right atrium immediately post injection of agitated saline

C. Contrast passing from RA to LA through PFO (red arrow)

D. Large amount of contrast in LA (red arrow)

(Courtesy of Thomas Binder, MD Univ Vienna)
Bilateral Carotid Dissection
TMB Type II due to retinal vascular insufficiency
TMB Type II

A sudden attack of temporary monocular visual loss, less rapid in onset and longer in duration (minutes to up to two hours) in comparison with Type I. Recovery also takes place gradually.
Visual loss characteristic of TBM Type II is a loss of contrast vision

Dazzle
“Over exposure”
Photographic negative
Flicker
Constricted peripheral vision
TMB Type II

Provoked by:

Systemic hypotension
Venous hypertension
Extra-cerebral steal
Low pressure retinopathy (Early)

Important compensatory mechanisms in the retinal circulation accompany a progressive reduction in retinal perfusion pressure. A low pressure retinopathy is characteristic.

- Venous distention
- Irregularity of the vein wall and leakage
- Blot hemorrhages and microaneurysms

Present in 20% cases ICA occlusion. Consider trans-cranial bypass.
Low pressure retinopathy (Late)

Signs of anterior segment ischemia usually co-exist:

- Rubeosis of the iris
- Neovascular changes in the anterior chamber
- Secondary glaucoma and cataract formation

Trans-cranial bypass too late
Low pressure retinopathy (Late)

Compensation becomes inadequate when both the external and internal carotid arteries are stenotic or occluded.

- Florid micro aneurysms
- Arterio-venous shunts
- Recurrent vitreous hemorrhage
- Retinal detachment and blindness
LCCA injection showed

Marked stenosis/subtotal occlusion LICA origin, lumen <1.0mm

Sluggish antegrade filling and delayed washout of proximal LICA

Minimal narrowing LECA origin

Extensive collateral reconstitution of supraorbital, supraocular orbital vessels with retrograde reconstitution of proximal LOA and cavernous LICA
In TMB Type I and Type II the etiology may be giant cell arteritis where there is a similar state of impaired retinal perfusion.
TEMPORAL ARTERY BIOPSY SPECIMEN (E70-717)

FIGURE NO. 1

AREAS OF GRANULOMATOUS INFLAMMATION

1.29mm 0.29mm 0.69mm 0.42mm 0.04mm
Figure 1. Giant-Cell Arteritis of the Temporal Artery. Panel A shows transmural inflammation of the temporal artery with granulomatous infiltrates in the media and giant cells at the media-intima border (hematoxylin and eosin, x 100). The lumen is partially occluded by intimal hyperplasia. Panel B shows a close-up view of a segment of the media with several multinucleated giant cells arranged adjacent to fragments of the internal elastic lamina (hematoxylin and eosin, x200).
GCA is a T-cell dependent disease

CD4+ T cells orchestrate the vasculitic process

T-cell activation requires the activation of specialized antigen-presenting cells, the dendritic cells

Antigens recognized by CD4+ T cells are unknown
TMB Type I or Type II may be the herald symptom of three common ocular strokes:

- Central retinal artery occlusion (CRAO)
- Branch retinal artery occlusion (BRAO)
- Anterior ischemic optic neuropathy (AION)
TMB Type III

Type III resembles Type II with less rapid onset and longer duration compared with Type I.

The mechanism is transient angiospasm.

In rare cases the retinal arteries appear narrow on funduscopy ± micro infarcts.

Migraine

Typical Features of Migraine-Related Transient Vision Loss

- Gradual onset and duration of up to 1 hour
- Positive visual phenomena
  - Photopsias
  - Scintillating scotoma
- Often followed by headache but may be acephalgic
- Associated photophobia and nausea
- Previous history of migraine

TMB Type IV

Idiopathic
Young women
Normal eye and fluorescein angiogram
Normal cardiac and vascular work up
± Antiphospholipid antibodies
Benign
Amaurosis Fugax Associated with Antiphospholipid Antibodies

Kathleen B. Digre, MD,*† F. Jane Durcan, MD,*† D. Ware Branch, MD,‡ Dan M. Jacobson, MD,§ Michael W. Varner, MD,‡ and J. Richard Baringer, MD*

In more than 50% of amaurosis fugax patients under 45 years of age no cause for the episodes of visual loss is identifiable. We have encountered 6 young adults (4 women and 2 men) with episodes of amaurosis fugax associated with elevated levels of antiphospholipid antibodies. Splinter hemorrhages of the nail beds were present in most patients. Treatment with antiplatelet medications and anticoagulants appeared to reduce the frequency of episodes and might prevent central retinal artery occlusions or stroke.

Patient 3
A 25-year-old woman with a history of spontaneous abortion experienced constriction of the visual field of the right eye at least 20 times each day. At other times she also experienced a unilateral central migrating scotoma lasting seconds to minutes at least 20 times each day. Clinical and laboratory findings in this patient are summarized in Tables 1 and 2.
Fig 1. Patient 1. Splinter hemorrhages were present on general physical examination.
<table>
<thead>
<tr>
<th>Type</th>
<th>1984</th>
<th>1985</th>
<th>1986</th>
<th>1987</th>
<th>Total</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>TMB n=33</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type I</td>
<td>24</td>
<td>31</td>
<td>34</td>
<td>42</td>
<td>(121)</td>
<td>80%</td>
</tr>
<tr>
<td>Emboli</td>
<td>1</td>
<td>8</td>
<td>12</td>
<td>3</td>
<td>(24)</td>
<td>19%</td>
</tr>
<tr>
<td>T. Arteritis</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>(12)</td>
<td>9%</td>
</tr>
<tr>
<td>ICA sten.</td>
<td>4</td>
<td>8</td>
<td>2</td>
<td>4</td>
<td>(13)</td>
<td>10%</td>
</tr>
<tr>
<td>Post. Endart.</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>(2)</td>
<td>1%</td>
</tr>
<tr>
<td>Fibromu. D.</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>(1)</td>
<td>-</td>
</tr>
<tr>
<td>Heart D.</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>(9)</td>
<td>7%</td>
</tr>
<tr>
<td>Type II</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>(10)</td>
<td>6%</td>
</tr>
<tr>
<td>Type III</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>(7)</td>
<td>4%</td>
</tr>
<tr>
<td>Type IV</td>
<td>4</td>
<td>2</td>
<td>5</td>
<td>3</td>
<td>(14)</td>
<td>9%</td>
</tr>
</tbody>
</table>
TMB Evaluation

Meticulous history
Ophthalmological exam / dilated funduscopy
Cardiac and carotid Bruits
  BP both arms rest and standing
  Heart rate / Holter / TEE
Sed rate / C-reactive protein / lipid panel / homocysteine / fasting glucose / HgA1c
± Temporal artery biopsy
Hypercoagulable workup
Carotid non-Invasive
Neuroimaging of TMB

Brain MRI (DWI/ADC)
MRA of the head and neck (fat saturation)
CT/CTA head and neck if TMB + signs of infarction (reformatted 3-d reconstruction)

* If cardiac embolic source or PFO suspected, may consider cardiac CTA as well (research)