The Optometrist's Role in Systemic Disease

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Course Goal

To provide clinically relevant information about the eye-body connection and the role of the optometrist in systemic disease.

Course Objectives

At the conclusion of this course, the attendee should be able to:

1. Describe relevant functional anatomy as pertains to common systemic conditions.
2. Explain methods to detect diabetes, impending stroke, neoplastic disease, IIH, and dermatologic conditions.
3. Detect ophthalmic features of cardiac disease, GCA, and carotid occlusive disease.
4. List common systemic and ocular malignancies.
5. Accurately diagnose and manage true papilledema.
6. Identify and manage ocular complications of dermatologic disease.
7. Identify relationships between systemic conditions and AMD.

Course Description

The eye is an extension of the brain. We perform dilated funduscopy to not only assess the integrity of the eye's posterior segment, but also to detect telltale signs of systemic disease. In this course, several patient cases are presented that illustrate ocular complications of a variety of systemic diseases. The symptoms, clinical features, pathogenesis, diagnostic workup, and management of these conditions are reviewed.

Course Outline

Part One: The Eye in Systemic Disease

Public Health Challenges and Epidemics

- Smoking
- Obesity
- Types 1 and 2 Diabetes
    - Children and adolescents
    - Adults
    - Diabetes in diverse populations
- Vision impairment and aging
    - Cataract
    - Age-related macular degeneration
        - Is AMD a systemic disease?
    - Glaucoma
    - Other optic neuropathies
    - Retinal vascular disease
        - RVO
        - RAO
        - OIS

Connective Tissue and Dermatologic Disorders

- Ankylosing Spondylitis
- Sjogren Syndrome
- ***Pseudoxanthoma elasticum***
- Ehlers Danlos syndrome
- Paget's Disease
- ***Rosacea***
- HLA B-27 associated conditions

Endocrine Disorders

- Diabetes Mellitus, Obesity, and the Metabolic Syndrome
    - Some important systemic effects of diabetes that affect **retinopathy** and increase the risk of Heart Attack
    - **Proteinuria**
        - First sign of renal disease
        - As nephropathy increases…the glomerular filtration rate falls
        - American Diabetes Association (ADA) recommends
        - yearly urinalysis
        - Random Spot Urine or 24-hour collection
        - **Normal <30**
        - **Microalbuminuria 30 mg – 299 mg, Albuminuria > 300 mg**

- **Anemia**
  - Gets more severe as renal disease worsens
  - Kidney production of **"Erythropoietin**" decrease, which means that less reaches the bone marrow and less red blood cells are made
  - Measured by Hematocrit (HCT) and hemoglobin levels in a CBC
  - If hemoglobin levels are less than 11g/dl = **anemia**
  - **Anemia** may actually be making the retinopathy worse!!!
  - Treat the patient with **Procrit** if patient is not on dialysis
  - Iron supplementation
  - Kidney Erythropoietin Bone Marrow RBC's
- **Hyperlipidemia**
  - Cholesterol and triglyceride healthy levels should be < 200 mg/dl
  - PCP should consider Lipitor if cholesterol high
- **Hypertension**
  - Target blood pressure for diabetics with nephropathy is 130 /80
  - ACE inhibitors should be given if blood pressure is high
  - ACE inhibitors are both renal-protective and anti-proteinuric
- **Hyperglycemia**
  - Induces vasoconstriction = kidney (glomerular) damage
  - The Hemoglobin A1C should be as close to 7 % as possible

Hematologic and Cardiovascular Disorders

- Giant Cell Arteritis
  - Acute painless vision loss (VA loss is usually permanent)
  - Pale swelling of the optic nerve head with flame shaped hemes
  - Central retinal artery occlusion may occur
  - Cranial nerve palsy (CN 3,4,6) may also be present, CWS
  - Possible association with "**Polymyalgia Rheumatica"** (PMR)
    - Stiffness in the neck, shoulder, and hip
    - 50 % of Giant Cell patients have PMR
    - Is there a link between GCA and PMR?
- Carotid Occlusive Disease
  - Ocular Ischemic Syndrome
    - **Hypoperfusion Retinopathy / Ocular Ischemic Syndrome:**
    - Usually unilateral but may be bilateral in 20% of cases
    - Males > Females by a 2 to 1 ratio
    - Dot and blot hemes / microanuerysms found only in the mid-peripheral retina = Hypoperfusion Retinopathy
    - When the above is associated with neovascularization of the Disc,
    - Retina, Iris or Angle = Ocular Ischemic Syndrome
  - **Pathogenesis: Ocular Ischemic Syndrome:**
    - Atheromatous ulceration and stenosis at the bifurcation of the
    - common carotid artery (90% occlusion has to be present)
  - **Symptoms: Ocular Ischemic Syndrome:**
    - Ocular and periorbital pain in **40%** of cases = **"Ocular Angina"**

        - Prolonged recovery of vision following exposure to bright light- known as "Light Induced Amaurosis"
        - Amaurosis Fugax (Transient Monocular Blindness) in 5% of cases
        - Transient Ischemic Attacks (TIA)
        - Vision Loss (90%) – Short Posterior Ciliary Arterial hypoperfusion
    - **Ocular Signs: Ocular Ischemic Syndrome:**
        - Dilated but not tortuous retinal veins
        - Retinal Hemorrhages in mid-peripheral retina (80%) of patients
        - Cotton Wool Spots (5%)
        - Neovascularization of the Disc (35%)
        - Neovascularization of the Retina (8%)
        - Rubeosis iridis (65%)
        - Uveitis – mild anterior (20%)
        - Emboli (retinal)
        - Lower IOP - initially
    - **Work Up:**
        - Carotid artery evaluation (Carotid – Duplex Scanning)
        - Possible MRA (Magnetic Resonance Angiography)
        - Cardiology work up (Echocardiogram) – Transesophageal
        - Lipid Panel
    - **Treatment:**
        - Consider carotid surgery if warranted (Endarterectomy)
        - Therapeutic approach – Aspirin (325 mg QD or BID)
        - Panretinal photocoagulation (PRP) if neovascularization
        - Stop smoking
    - **Important Note:**
        - Leading cause of death = **Ischemic heart disease**
        - Second leading cause of death = **Stroke**

Part 2: Is AMD a Systemic Disease?

I. Introduction

A. AMD Defined
1. Evidence for AMD as a systemic condition
2. Systemic treatments and strategies for AMD prevention and Treatment.

B. Co-management and Interprofessional Care Defined
1. Co-management- a relationship between practitioners for shared responsibility in patient care.
    a. Patient must consent
    b. Providers must agree to share patient care.
2. Interprofessional Practice/Care- the provision of comprehensive health services to patients by multiple caregivers who work collaboratively to deliver quality care within and across settings.
3. General Guidelines for Co-management and Referral

II. Epidemiology

A. Statement of the Problem
1. Prevalence of Ocular Diseases
B. The Burden of Disease
C. The Burden of Treatment

III. Systemic and Other Aspects of Age-related Macular Degeneration

A. Age Related Eye Disease Study estimated a prevalence of more than 8 million individuals in the US with at least "intermediate" AMD in one eye who are at risk for "advanced" AMD.
1. AMD has a genetic component but the disease is "multifactorial".
2. Environmental, dietary, medical and lifestyle factors are influential.

B. Risk Factors
1. Cigarette Smoking – most important avoidable risk factor
2. Age – most significant risk factor (> 65 years old)
3. Positive family history of AMD
4. Hypertension
5. High levels of LDL and low levels of HDL cholesterol
6. Diet rich in polyunsaturated fats
7. Female gender
8. Cardiovascular disease
9. Hyperopia
10. Blue iris color
11. Increased C-reactive protein
12. Increased white blood cell count
13. Obesity (higher body mass index)
14. Race: Whites > Hispanics > Blacks
15. Low Macular Pigment Optical Density (MPOD)
16. Poor dark adaptation

C. Systemic Wellness, Diet and AMD

1. Dietary "Ancillary Study" within the multi-center "Eye Disease Case-Control Study"

2. Evaluated the relationships between the intake of carotenoids and Vitamins A, C and E and the risk of ARMD

3. Results showed that a higher dietary intake of carotenoids was associated with a lower risk for ARMD

4. Also showed that the carotenoids, lutein and zeaxanthin were most strongly associated with this reduced risk

5. The National Health and Nutrition Examination Survey

a. Found that higher levels of lutein and Zeazanthin were related to lower odds for pigment abnormalities (an early sign of AMD)

6. Lutein and Zeaxanthin Appear naturally in the macula
   a. Are an essential dietary carotenoid (yellow to red pigments found in plants)
   b. Functions as a natural sunscreen (decreases blue light scatter)
   c. Provides antioxidant defense against free radicals
   d. May improve visual function

D. Available technologies for early detection and monitoring of AMD

- Macular Pigment Optical Density (MPOD)
- Optical Coherence Tomography (OCT/OCTA)
- Dark Adaptation

E. Systemic Treatment, Management, and Co-management of Dry AMD

F. Systemic Treatment, Management, and Co-management of Wet AMD

Part 3: Other Ocular Manifestations of Systemic Disease

A. Diabetic Retinopathy: Systemic Treatment, Management, and Co-management

B. Retinal Vascular Occlusions: Systemic Treatment, Management, and Co-management

a. RAO
b. RVO

C. Neoplastic Disease

a. Primary Ocular Cancers
b. Metastatic Cancers to the Eye
   - Most common primary sites

D. True Papilledema in IIH

OCT findings associated with true papilledema:
•increased nerve fiber layer thickness
•elevation of the optic nerve head
•maintenance of the central cup*
•a subretinal hyporeflective space between the photoreceptor layer

and RPE
• recumbent “lazy V”
•an inward deviation of the RPE/Bruchs complex

Empty Sella
• Characterized by enlargement or malformation of the sella turcica.
• In empty sella syndrome, the sella turcica is either partially filled with cerebrospinal fluid and a very small associated pituitary gland lying in the floor of the sella (partially empty sella) or
• completely filled with CSF with no visualized pituitary gland (completely empty sella).
Neuroimaging
• MRI with and w/o contrast of brain, orbits, canals, fossae
• MRV to r/o cerebral venous sinus thrombosis
••
Idiopathic Intracranial Hypertension (IIH)
–Increased intracranial pressure without an identifiable cause

Idiopathic Intracranial Hypertension (IIH)
–Increased intracranial pressure without an identifiable cause
–Young, obese females are at risk
–Tx: po Diamox, weight management

Papilledema vs Pseudopapilledema A/Bscan Echography

Key Points
•An elevated optic nerve head may be edematous or non-edematous.
•True optic disc edema has many causes.
•Not all disc edema is papilledema!!
•Clinicians must be adept at examining the optic nerve and associated structures.
•Physical examination together with patient history and other specialized testing (such as perimetry, OCT, echography, neuroimaging) should help differentiate true disc edema from pseudo-edema.

Thank you!

Joe, Anthony, and Carlo