

Inflamma-story: The Role Inflammation Plays in Dry Eye Disease

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One Hour COPE

Category: Anterior Segment Disease

Course description:

Dry eye disease is a chronic and, possibly, progressive inflammatory condition. With millions of symptomatic patients, proper diagnosis and management of inflammation is integral in treatment. This course will discuss the innovative diagnostic tools and therapies emerging in clinical care and how they are applied to benefit patient outcomes.

Course Objectives:

- To educate attendees on the pathophysiology of inflammation in dry eye disease
- To discuss diagnostic tools to help identify inflammation in a clinical setting
- To identify established and emerging ophthalmic treatment options used to mitigate inflammation in the dry eye patient
- To discuss the interaction of the skin and dermatological conditions with inflammation

Outline:

- I. Dry eye disease (DED) origins
 - a. Evaporative defined
 - i. Most common form of DED
 - ii. Inflammation, microbial overgrowth and associated skin disorders play a role
 - b. Aqueous-deficient defined
- II. Inflammation in DED
 - a. Heterogeneous, combined affect of:
 - i. Eyelid inflammation
 - ii. Conjunctival inflammation
 - iii. Corneal damage

- iv. Microbiological changes
 - v. Tear Instability related DED
- III. The interaction of DED and Meibomian Gland Dysfunction (MGD)
 - a. MGD
 - i. Self-stimulated by microbiological changes
 - 1. Increasing the melting point of meibum
 - 2. Causing blockage
 - b. DED
 - i. MGD-related tear film instability provides entry point for DED
 - ii. Hyperosmolarity
 - iii. Inflammation
- IV. Skin diseases (ocular rosacea) and MGD
 - a. 90% of patients with ocular rosacea show eyelid changes similar to those with MGD
 - b. Absence of normal lid meibum, entry point for DED in rosacea patients
 - i. Increase in lipid-deficiency
 - ii. Increased tear evaporation
 - iii. Hyperosmolarity
 - iv. Inflammation
 - c. The “missing link” between eyelid inflammation and lacrimal effects
 - i. Exposure of ocular surface epithelia to desiccating stress– resulted in release of cornified epithelial precursors by the ocular epithelium

- ii. Causing further blockage of meibomian glands and loss of goblet cells

V. Diagnosis of inflammation in the tear film

- a. Imaging devices

- i. In vivo confocal microscopy (IVCM)

- ii. Optical coherence tomography (OCT)

- iii. Keratography

- b. Inflammatory testing

- i. Matrix metalloproteinase-9 (MMP-9)

- 1. Inflammatory biomarker

- 2. Conditions with elevated MMP-9

- a. DED/MGD

- b. Corneal ulcers

- c. Sjögren syndrome

- d. Ocular rosacea

- 3. Ranges of MMP-9 in the tear film

- a. >40ng/ml for positive result

- b. Correlation with other diagnostic DED tests

- i. Fluorescein tear break-up testing

- ii. Fluorescein staining

- iii. Ocular surface surveys

- ii. Inflammadry testing

- iii. Osmolarity/MMP-9 platform testing

c. Treatment of inflammation in DED/MGD

i. Ophthalmic medications

1. Cyclosporine

a. Cyclosporine, 0.05%

i. Study data

1. Measurement of aqueous volume

b. Cyclosporine, 0.09%

i. Study data

1. Measurement of aqueous volume

2. Lifitegrast, 5%

a. Indication

b. Mechanism of action

c. Study data

i. Endpoints (2-week, 6-week, 12-week)

ii. Symptom results

1. Eye dryness score (EDS)

iii. Sign results

1. Inferior corneal staining

3. Azithromycin

a. Clinical uses

i. Dual action of anti-inflammatory and anti-microbial

b. Noted as "off-label" use

ii. Dermatological treatment

1. Intense Pulse Light (IPL)

- a. Proposed mechanism of action
- b. Study data
- c. Potential benefits
 - i. Ocular
 - ii. Dermatological
- d. Potential adverse events or risk
- e. Patient selection