A BRIEF OVERVIEW PRESENTED AT DRY EYE UNIVERSITY OF INFLAMMATION IN CHRONIC DRY EYE DISEASE

Jerry Robben, OD
Chief Optometrist and Director of Clinical Research at Bowden Eye & Associates in Jacksonville, FL. Adjunct Clinical Professor at Midwestern University, College of Optometry in Glendale, AZ.

Corresponding Author: jrobben@bowdeneye.com

Published, April 16, 2020.

At Dry Eye University (DEU) we welcome eye care providers (ECP), their support staff and administration of all experience levels in relation to dry eye expertise. One goal we have is to create a more unified approach in treating dry eye disease by all ECPs. The recognition of inflammation as a core mechanism in dry eye by all is a part of that goal. We also want to educate on the importance, we feel, of addressing that inflammation as a first line treatment. At our practice, Bowden Eye & Associates, our dry eye standard of care indicates that once we have diagnosed a patient with dry eye disease, we need to prescribe some level of anti-inflammatory treatment as a first-line treatment, along with a customized regimen to support the dry eye patient and hope to gain control of their disease process.

We make this point numerous times throughout our DEU programs because we know of a long-standing trend for ECPs to hold anti-inflammatory treatments until patients become more moderate or even severe, sometimes after long periods of following these patients (and watching them worsen).

Just look at all of the previous working definitions of dry eye disease. Inflammation is a common part of every major definition that we have used to understand dry eye disease.

Pflugfelder et al. in 2004 “Dry Eye is a disorder whereby dysfunction of the lacrimal functional unit causes an unstable tear film, which in turn promotes ocular surface inflammation, epithelial disease and symptoms of discomfort.”

DEWS 2007 “Dry Eye is a multifactorial disease of the tears and ocular surface that results in discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by inflammation of the ocular surface.”

DEWS II 2017 “Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.”

With this very obvious fact about inflammation and dry eye, why is it frequently ignored by physicians treating known dry eye patients? Possibly it is because basic understanding of inflammation is overlooked. At Dry Eye University we review inflammation is a very basic sense to simply illustrate the importance of treating it as early as possible in our dry eye patients.

This simple review starts with a discussion about the normal inflammatory cascade as stated in the book Fundamentals of Inflammation by Peter Ward.

“Inflammation is a beneficial host response to a foreign challenge or tissue injury that leads to the restoration of tissue structure and function. It is a reaction of the microcirculation characterized by the movement of serum...
proteins and leukocytes from blood to the extravascular tissue. This movement is regulated by chemotactic mediators that result in the classic signs of inflammation – heat, redness, swelling, pain and loss of tissue function. Then local vasodilation increases blood flow to the area and the blood vessels become more permeable which releases the plasma proteins into the tissue. Adhesion molecule expression is upregulated and the inflammation site releases chemotactic factors – this facilitates circulating cells to adhere to the vascular endothelium to then migrate into the affected area. This all leads to the accumulation of polymorphonuclear leukocytes (PMNs) in the inflamed area, which later are replaced by monocytes. The monocytes differentiate into macrophages which are phagocytic cells that ingest foreign material and cell debris and release enzymes that also attack any foreign organism. And, finally the injurious stimulus is cleared and normal tissue structure and function is restored.4

But what if all of this doesn’t turn off? What if the stimulus that started it, continues to be present? What if the injury never heals? Then we get chronic inflammation, and once that happens, the inflammatory cascade that is designed to heal us, becomes the disease itself. The ongoing inflammatory cascade and accumulation of inflammatory cells begins to damage the tissue and begins to cause loss of organ function.4

This is the fundamental understanding that we want to highlight with this discussion. Inflammation is a common enemy of any chronic inflammatory disease. Dry eye is no different. Once this diagnosis is made, then one of the first objectives is to address and control the chronic inflammation appropriately. Doing otherwise, and not addressing it, will likely result in prolonged uncontrolled disease and worsening of the patient’s condition.

REFERENCES