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Dry Eye Symptoms in Wet Eyes

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Opinion Article

Evaporation is a ubiquitous threat to the quality of the tear layer surface that serves as the most powerful focusing lens of our eyes. Sustaining this function in face of the omnipresent threat of its evaporative degradation was a major obstacle to the migration of our remote ancestors from their water habitat to land. The development of a tear thickness-monitoring alarm/tear secretion system was a key factor in the evolution of our species.

Conventional wisdom attributes the dry eye-like symptoms of eyes with adequate tears to their accelerated evaporation. However, observations that the volume of tears is too generous and the Meibomian glands are normal in some of these patients question this belief and suggest the participation of an important and still unidentified pathogenic mechanism.

Sensations of dry eye are the alarm signal of pending or actual pre-corneal tear film breakup that causes a sudden loss of functional vision when it occurs. The rate of tear secretion required to sustain the optical quality of the tear surface in the face of a rapidly changing external environment is controlled by specialized sensors in the TRMP8 channels embedded in the terminals of corneal nerves and which are sensitive to increased rates of temperature drop [1] such as caused by tear evaporation. This rate increases as the tear layer become thinner until a threshold is breached thereby triggering their activation that leads to the increased secretion of tears. Although these events normally occur at an unconscious level when the thickness of the tear film falls below a certain threshold level the resulting increase in the rate of cooling of the corneal surface breaches the threshold of these sensors and trigger the dry eye alarm (symptoms of dry eye) warning us that our functional vision is at risk. In this context, I propose that the inappropriate dry eye symptoms of eyes with normally adequate tears are caused by a pathological increase in the sensitivity of these sensors and that accelerated tear evaporation, when it occurs, is a secondary contributing factor. This theory poses the question of the likely cause of because the proposed increased sensitivity of these sensors. I suggest that is an unrecognized corneal neuropathy.

Inappropriate dry eye symptoms are also a common feature of the ocular neuropathic pain syndrome [2] that can also cause

sensations of burning, stabbing, cutting, pins and needles and/or pressure perceived as originating in the orbits, face, ears, jaw and are commonly associated with headaches. Moreover, I suggest that the striking disparity between the intensity of these symptoms and paucity of causal signs typical of this disorder is the result of the malfunctions in the sensors of the corneal nerve terminals being opaque to current methods of study. Although this pain syndrome can occur spontaneously or in association with diseases such as Sjogren's syndrome and fibromyalgia it is often preceded by ocular trauma, most commonly LASIK and PRK. (I refer the readers to a paper posted on the website of the non-profit Boston Eye Pain Foundation that was rejected by several peer-reviewed journals prior to the publication of papers that introduced the concept of ocular neuropathic pain being a disease in its own right [3].

The striking disparity between symptoms and signs has led us to minimize the complaints of those patients in whom the paucity of signs belies the severity of their disability as is characteristic of patients who experience dry eye symptoms despite the presence of adequate tears. And this also could explain why the neuropathic had not been previously considered. If the suggested hypersensitivity mechanism is validated by future studies it could open the door to the development of long-needed more effective therapeutic strategies.

In the absence of better treatments the enormous personal, societal and economic costs of this disease will continue to escalate. Hopefully, the current lack of effective therapeutic options and logic of this neuropathic paradigm will expedite the studies required to validate its authenticity that if they do, could open a door to the development of substantially superior treatment strategies and perhaps even eventually cures for many of the victims of this disease.

References

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