A woman with dry eyes exacerbated by driving

Commentary by
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A 72-year-old woman with dry eye syndrome is finding her symptoms are markedly exacerbated by driving. How can she be helped to ensure she can keep driving?

Case scenario

Patricia is a 72-year-old woman who is very distressed about her recurring eye pain. She has had a complete eye check performed by an ophthalmologist, and has been reassured that her eyes are healthy apart from her having the common condition of dry eyes. She had been advised to use artificial tears as needed.

Patricia reports, however, that recently she has been developing severe pain and burning in both eyes whenever she drives her car. She develops such eye discomfort that she needs to stop after 10 to 15 minutes of driving to use lubricating eye drops, which then give her some temporary relief. She has to pull over repeatedly to administer the drops during longer trips. This severe discomfort does not occur when she is a passenger in the car or in any other setting. She has found no correlation with her car heating, air conditioning or window airflow.

What is causing Patricia's marked eye discomfort while driving and what can be done to help her?

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Commentary

From this history, Patricia's symptoms are almost certainly a consequence of dry eye syndrome (DES).[1,2] The notion that 'her eyes are healthy apart from … dry eyes' trivialises what really is a serious condition, sometimes associated with considerable disability. It also highlights the fact that there is no one single test that provides this diagnosis with certainty. Typically, several tests are used to make a clinical decision, to grade disease severity and then to recommend effective treatment. Apart from causing difficulties with driving, dry eyes can have an impact on a patient's quality of life as significant as that of moderate angina. Such patients may suffer from depression, can be unhappy and experience a deterioration in their mental health.[3-6]

Patricia's eyes are likely to be unhealthy. The International Dry Eye WorkShop (2007) defined dry eye as 'a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface'.[7] As the tear film is the first refractive surface of the eye, disturbances can reduce visual quality, acuity and field. In a visually demanding situation such as driving, akin to staring at something that you do not want to miss (such as a crucial part of a computer game), even a normal tear film is stressed. It has since been shown that the entire cornea can be profoundly affected by this disease, and there have been advances in understanding its effects on corneal innervation and pain.[8-10] The corneal nerve density is 300 to 600 times that of the skin – perhaps the most densely innervated tissue in the body.[10,11] Although issues relating to pain in DES are complex, several studies have shown reduced corneal nerve density and this correlates with ocular surface staining, consistent with the known trophic effects of corneal nerves.[10,12] In some studies, ocular surface disease correlates with reduced corneal sensation and fewer dry eye symptoms – typically, such patients present with or

complain of reduced vision.[13] On the other hand, DES can be associated with corneal hyperaesthesia – so called 'pain without stain' – and Patricia may fit into this category.[14,15]

Patricia is at risk of having dry eye by virtue of her age and sex, but other risk factors should be assessed. Systemic medications (the 'anti' drugs – antihypertensive agents, antidepressants, antihistamines), alcohol intake, smoking, obesity, the metabolic syndrome, vitamin D deficiency, migraine and a range of autoimmune diseases can be associated with DES.[1,16-18] Specific eye factors such as associated blepharitis (with or without rosacea), previous eye surgery (refractive, cataract, blepharoplasty) and eyelid tattoos (not uncommon in this setting) may play a role.[2,19] If possible, such factors should be dealt with while treating the ocular surface. Omega-3 fatty acid supplementation can be helpful.[20] There is no universally accepted dosing schedule for omega-3 fatty acids in patients with DES, and various plant and marine sources have been tried. An omega-3 fatty acid dose of 500 mg twice daily (containing 325 mg eicosapentaenoic acid and 175 mg docosahexaenoic acid) has been shown to be effective.[21]

A number of environmental factors can play a role in the symptoms associated with DES, and driving is specifically problematic, providing a 'stress test' for the ocular surface.[1] The effect of DES on driving has been recognised, with patients with dry eye reporting more problems with driving than control subjects.[22,23] A driving simulator study showed that patients with DES had increased average response times and a significant increase in percentage of missed targets compared with control subjects.[24] The performance of patients with DES was worse in specific driving situations, such as at crossroad and roundabout approaches. Response times were correlated with ocular aberrations (increased in patients with DES) and a DES questionnaire.

Blink rates appear to be higher in patients with DES.[25] It has also been shown that patients with DES spend more time with their eyes closed (4.5%) when watching a video than normal subjects (0.7%), which might play a role in the visual function decay associated with increased blink rates.[26] The act of gazing, as occurs during driving, suppresses the blink reflex, contributing to desiccation of the ocular surface, which has been directly demonstrated.[25] Interestingly, blink rates when driving decrease with increased driving speeds, at least in normal subjects, so it is possible that Patricia might have greater difficulty at high speeds. Similar problems are encountered with dynamic visual display terminal tasks, such as playing computer games.[27] It was found that blink amplitude and rate and tear film stability are compromised during these tasks, which was thought to have a negative effect on both the cognitive aspects of the task and the rate at which new visual information is presented. There may be similar factors at play during driving, which would exacerbate dry eye symptoms.

In general, the usual treatment options for patients with DES of lubrication, tear conservation and lifestyle changes are palliative rather than addressing the underlying pathophysiology. Although the mainstay of treatment for patients with DES is ocular lubrication, a large US survey of patients found that fewer than one in eight patients reported significant success with the three most common treatment options (artificial tears, oral antibiotics and topical corticosteroids).[28] Newer lubricant preparations containing lipid (to retard tear film evaporation) or hyaluronic acid and osmoprotectants may represent an advance.[29,30] Lacrimal punctal occlusion has been shown to improve functional visual acuity.[31]

However, recognition of the crucial role of ocular surface inflammation in DES and its management with topical cyclosporin (0.05% twice daily) has been a major advance in treating patients such as Patricia.[1] There is a response rate of 70 to 80% in patients with mild to moderate disease and about 60% in those with severe disease.[32] The effect is not immediate but may take a few months, during which time low-dose nonpreserved corticosteroid eye drops can play a role. Topical cyclosporin has also been shown to increase tear production, improve ocular surface health and reduce reported blurred vision.[33,34]

If nonpreserved lubricants are ineffective, autologous serum eye drops or lacrimal punctum plugs can be used. Typically, only one punctum per eye is blocked, permitting some egress of tears and their role in 'washing' the ocular surface. Temporary plugs can be used as a trial before the more permanent variety is fitted.

It has recently been shown that a factor in a positive response to treatment of DES is the presence of near-normal corneal innervation.[10] It is possible that early, aggressive treatment (before loss of corneal nerves) may result in a better response. It is probable that Patricia is in an early phase of DES, when environmental factors are not playing a major role, and she can be reassured that treatment is likely to alleviate her symptoms. MT

References

A list of references is included in the website version (www.medicinetoday.com.au) and the iPad app version of this article.

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