CORNEAL VASCULARISATION AND CONTACT LENSES

VASCULARIZACIÓN CORNEAL Y LENTES DE CONTACTO

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The cornea is avascular for the simple reason that to be otherwise would compromise its role as the primary refracting surface of the eye. As a result, situations that may cause vascularisation of this beautifully transparent tissue are energetically avoided. Visual disturbance caused by vascular encroachment upon the central cornea region is, perhaps, the most obvious threat. However, corneal vascularisation (CV) has other potentially damaging consequences including jeopardizing the normally immune privileged status of the anterior chamber (1) and increasing the risk of graft rejection (2,3).

For the general population, the most likely situation in which CV will be encountered is in association with contact lenses. Although prevalence estimates are quite variable, the suggestion is that between a tenth and a third of all cases of CV have contact lens involvement. Not all lenses are equal in this respect however. Prevalence figures tend to hide the fact that rigid gas permeable (RGP) lenses are much less likely to be associated with CV than are soft contact lenses (SCL). Although the mechanisms whereby contact lenses cause CV are not completely understood, the divergent responses to the various lens types are instructive and consideration of the functional differences between these modalities can provide valuable aetiological insight.

Typical RGP diameters are in the region of 9-10mm and consequently cover only the central portion of the cornea during wear. Conversely, SCLs are substantially larger, at around 13-15 mm, and cover the entire cornea as well as the limbus and some of the surrounding peri-limbal conjunctiva. The general effect of this additional coverage with SCLs is to reduce access of the underlying tissues to oxygen from the atmosphere and that dissolved in the tear film. As a result, the peripheral cornea and limbus are likely to experience some degree of hypoxia. An early manifestation of this is hyperaemia within the limbal vessels, a response that has been recognized for many years during SCL wear and one that is directly associated with the hypoxia they produce (4). Although there is debate about whether peripheral hypoxia is a sufficient stimulus in itself to cause CV, the associated short term vascular changes are apparently identical to those seen where CV does eventually occur.

One other key aspect of lens wear that impacts CV is the mode of use. Wearing lenses for long periods of time, and particularly during periods of sleep, i.e. extended or continuous wear, carries a higher risk than the conventional daily wear format where lenses are removed prior to eye closure. Conditions in the closed eye are indicative of a state of sub clinical inflammation with huge increases in polymorphonuclear leukocyte recruitment and the upregulation of several factors that have potentially angiogenic properties (5). Under normal circumstances these are balanced by a complementary upregulation of angiostatic factors, thus maintaining the status quo. Contact lens wear appears to carry the potential to alter this balance and again the increased hypoxic load may be the key factor. During sleep, the oxygen tension at the front surface of the contact lens reduces from the 155 mmHg available in the atmosphere to that provided by the vessels of the palpebral conjunctiva, i.e. about 55 mmHg. Placing a contact lens between the closed eyelid and the cornea potentially further restricts this already reduced oxygen supply.

Until recently soft contact lens materials had insufficient capacity to transmit physiologically adequate levels of oxygen to the ocular surface over the full range of circumstances encountered during

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wear. Hypoxic sequelae, including CV, have thus been commonly encountered complications of SCL wear. When faced with corneal vascularisation induced by SCLs, the strategy of the clinician must be to reduce the hypoxic load. Traditionally this has meant either reducing wearing times or increasing oxygenation at the ocular surface by reducing lens thickness and/or increasing the water content of the lens. Of course the option of switching to an RGP lens is always available, though this is seldom popular with the wearer because of the greater initial discomfort and longer adaptation period involved.

Recently, a substantial new alternative has been provided by the emergence of silicone hydrogel materials. These new polymers allow the fabrication of contact lenses with the same dimensions and comfort levels as traditional SCLs but with very much higher oxygen transmission properties. Use of these lenses makes it possible to reduce hypoxia during wear to levels that, in many cases, approximate the non-wearing situation. Early indications are that these materials can significantly reduce the occurrence of CV during SCL wear (6).

While contact lens associated CV would benefit greatly from the elimination of hypoxia, it would not be reasonable to assume that this is the only cause. Poorly designed or badly fitted lenses can cause direct mechanical injury to the ocular surface or, if coupled with poor biocompatibility, severe tear film disruption. If sufficiently prolonged, the damage these events create may be sufficient to initiate the events of the angiogenic cascade. It is also worth remembering that while the majority of contact lens wearers do so for cosmetic reasons there is a substantial group who have therapeutic needs. These individuals typically require special lenses. For example, conditions such as keratoconus, high myopia or irregular cornea may call for scleral lenses and due to their large size and thickness these lens types may present a significant physiological challenge to the ocular surface, as well as to the fitter. It is not surprising then, that these groups experience CV more regularly or that its severity is relatively higher than in the cosmetic population. Once again however improvements do appear to be obtainable where it is appropriate to utilize materials of higher oxygen transmissibility (7), although the scope for doing so is currently relatively limited. Hopefully continued development in terms of material properties will also be able to benefit this group in the near future.

REFERENCES

4. Papas EB. The role of hypoxia in the limbal vascular response to soft contact lens wear. Eye Contact Lens 2003; 29: S72-S74.