

STUDIES IN THE AETIOLOGY OF PTERYGIUM

Part 3—A Case of Artificial Ultraviolet Light—Induced Pterygium

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SUMMARY: An unusual case of bilateral pterygium is described and the aetiology is discussed.

The facts described are related to those presented in the two previous papers and a case is argued that the damaging effect of ultraviolet light (U-VL) from solar radiation is the main aetiological factor.

Introduction

Although never convincingly explained, it is a platitude that pterygium occurs on the nasal side of the cornea rather than on the temporal, and almost never temporally unless the nasal sites are already involved. It is therefore of interest to record a case in which pterygium occurred nasally in one eye and temporally in the other. The history contains an extremely interesting and highly suggestive probable aetiological factor.

Case history

A 20-year-old Guyanese Corporal in the British Army reported to his medical officer complaining of irritation in both eyes for one week and was referred for specialist opinion when he was seen to have pterygia in both eyes. Examination showed that he had a temporal-sided pterygium on the left eye and a nasal-sided pterygium on the right with no sign of pterygium in the other two sites. The pterygium on the right eye showed activity, with pre-apical islets of opacity, translimbal vascularisation and pigment granules at the level of Bowman's membrane. It had progressed 1.5 mm over the limbus. On the left eye, temporally, was a typical true pterygium which had transgressed the cornea by 2.0 mm and was well vascularised. No pigmentary changes were seen. Neither side showed fluorescein staining, even under ultraviolet light (U-VL).

The subject was born in British Guiana and lived there until he was almost 16 years old when he went to England. Guiana lies 5° North of the Equator and has a sea-coast and may be expected to produce a fair incidence of pterygium. For a period of 9 months, from the age of 16 years and 9 months he was employed as an electric arc spot welding machine operator, making metal cases for electric meters. Although he was issued with protective goggles he seldom wore these, indeed only "when the inspector was coming". The goggles were uncomfortable and he could not see properly while wearing them.

Being left-handed, he always stood to the right of the welding machine, but had to bend over close to the work each time he made a weld in order to check that the electrodes were properly applied. (Improper application usually led to a burn-hole in the sheet metal and a spoiled piece). He estimated that his face was within 30 cm of the arc at all times, but often much nearer. He was employed, full-time, on the spot-welding machines from 0800 to 1700 hours on three days of the week, and from 0800 to 2000 hours on two days a week.

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After leaving this employment he joined the Army, in February 1966, and became a seaman in the Royal Corps of Transport. He was posted to Singapore in September 1968 and, when first seen, had been employed for a year on water transport duties.

He insists that he was quite unaware of any eye trouble until a week before reporting to his doctor and he had not noticed his pterygia. His only complaint was of irritation and "foreign body" sensation.

Discussion

That chronic exposure to solar radiation is an important factor in the aetiology of pterygium, is almost undeniable: the evidence is now very strong. Whether the ultra-violet part of the spectrum of sunlight is the sole, or even the main, cause, is however, another question. There is an obvious identity between solar keratopathy (snow blindness) and the effects of exposure to radiation from the electric welding or cutting arc. Indeed, the clinical manifestations may be indistinguishable. Perhaps not so obvious is the identity with the condition of low-grade solar keratoconjunctivopathy so familiar to those of us who work in tropical areas dealing with Caucasian patients exposed to the mid-day sun. As one watches these patients' pingueculas gradually transgress the limbus and notes the punctate corneal epithelial staining and the impressive conjunctival injection, little exercise of imagination is necessary to read the picture as a mild form of "welders' eye".

Urbach, Davies and Forbes (1965) investigating skin carcinoma, studied the relative dose-rates of solar U-VL on different parts of the face and showed that this was considerably influenced by the anatomical contours. Sevel and Sealy (1968) whose work in Groote Schuur involves considerable preoccupation with the effects of U-VL on the eye, have pointed out that sunlight-induced blepharospasm leads to covering of the temporal interpalpebral conjunctiva, leaving the nasal area exposed. It would be interesting to check whether patients who develop temporal pterygia, secondary to nasal-sided lesions are anatomically unable to avail themselves of this partial protection.

This case is particularly interesting because of the probability that the pterygia were caused, or at least precipitated, by the repeated exposure to welding flash. The fact that the subject is left-handed and always stood to the right of the machine, taken in conjunction with the fact that his pterygia are on the temporal side of the left eye and the nasal side of the right, strongly suggests a causal link.

The life history, however, is one of considerable total U-VL radiation dosage and it is probable that the industrial episode, although important, was contributory or precipitative rather than the sole aetiological factor. It is noteworthy that symptoms developed after a year of exposure to marine duties in tropical waters. Eye irritation from solar conjunctivopathy is, in fact, a commonplace among the personnel of the Water Transport Coy RCT., Singapore: considerable numbers of these men eventually find their way to the Eye Department and a number of pterygia, in Europeans, have arisen from this source. Presumably those employed on small craft are exposed to additional ambient radiation by reflection from the water.

How is it possible to tie up all the facts presented in this paper and the two previous papers (this issue pages 117 to 125) into a convincing hypothesis on aetiology? Before attempting to do so it is necessary to refer, briefly, to established knowledge of the histopathology of pterygium. A good recent account is given by Takebayashi (1966).

The earliest observable change ahead of the apex of the growing pterygium is a cellular invasion of the space between the basement membrane of the corneal epithelium and the underlying Bowman's membrane. Subsequently, the basement membrane thins in places, fragments and finally disappears. Then the same fate overtakes Bowman's membrane. Changes in the stroma of the cornea, deep to Bowman's membrane, are negligible. The epithelium of the neck of the pterygium is considerably proliferated and contains many goblet cells. Subepithelially, the connective tissue contains areas of degeneration and there is considerable neo-vascularisation and an increase in elastic fibres.

I think the considerable volume of information we now have on pterygium may be incorporated into the following scheme, which is an attempt to suggest the mechanism of pterygium formation.

Ultraviolet light of wavelength between 290 and 300 nanometres is able to penetrate the atmosphere around mid-day in the peri-equatorial zone in sufficient quantity to cause damage to the epithelia of cornea and conjunctiva by radiation effects on deoxyribonucleic acid (DNA) molecules. This occurs only in the interpalpebral area because the rest of the corneal and conjunctival epithelium is protected by the lids. Because the cornea is avascular it cannot respond by inflammation, but the conjunctiva does so, producing tropical pinguecula. The conjunctival manifestations occur mainly on the nasal side because bright light blepharospasm is more effective in protecting the temporal side of the conjunctiva than the nasal.

The effect of U-VL on the cornea is to lead to patchy destruction of the basement membrane of the epithelium and Bowman's membrane. Possibly, antibody effects may sensitise to later exposure. Localised destruction of Bowman's membrane produces the pre-ptyerygial islets of opacity readily visible on bio-microscopy. It also leads to the establishment of non-epithelialised areas on the cornea, which can be observed as punctate, fluorescein-staining patches.

The stage is now set for the migration of conjunctival epithelium across the limbus; and the start of a pterygium. This does not necessarily occur, however. Much depends on the state of the adjoining conjunctiva. Being better nourished, the conjunctival epithelium is better able to resist destruction than is the corneal epithelium. Its response to chronic irritation is to proliferate and it is possible that U-VL irritation in "natural" doses, alone, is insufficient to lead to trans-limbal spread. Other factors, contributing to conjunctival irritability may be necessary. U-VL, in excessive dosage, is, however, almost certainly capable of causing pterygium.

Exuberant conjunctival epithelium can readily gain access to the cornea via de-epithelialised patches lying on or near the limbus. De-epithelialised areas more centrally placed cannot initially progress to pterygium because they are separated from conjunctival epithelium by a barrier of largely intact corneal epithelium. Once the conjunctivocorneal epithelial bridge is established, sub-conjunctival connective tissue spread can occur and the pterygium thickens and assumes its classical form. Now, the sub-ptyerygial cornea is protected from U-VL but corneal basement membrane ahead of the apex of the lesion is not protected, and the process may thus be propagated towards the centre of the cornea.

Corneal epithelial slide, to cover superficial defects, is a well-known mechanism. This will operate to deal with any centrally-placed patches of de-epithelialisation. In

the case of peripherally-placed patches, however, the situation becomes a race between corneal epithelial slide and conjunctival epithelial transgression. Sometimes the one wins, sometimes the other. Presumably the outcome depends on the relative viability of the two epithelia.

It is difficult to explain why pterygium never grows beyond the centre of the cornea, but, bearing in mind that pterygial size and frequency are reciprocal parameters, this may simply be another way of saying that pterygia never grow large enough to reach beyond the corneal mid-point.

In view of the evidence now linking pterygium with tropical U-VL it would seem to be worth-while promulgating the dangers of repeated exposure to noon-day sun in tropical areas. In particular, deliberate exposure in sunbathing, during the period of maximum ambience of damaging radiation, is to be deplored, not only because of the now fully established dangers of skin carcinoma, but also because of the possibility of

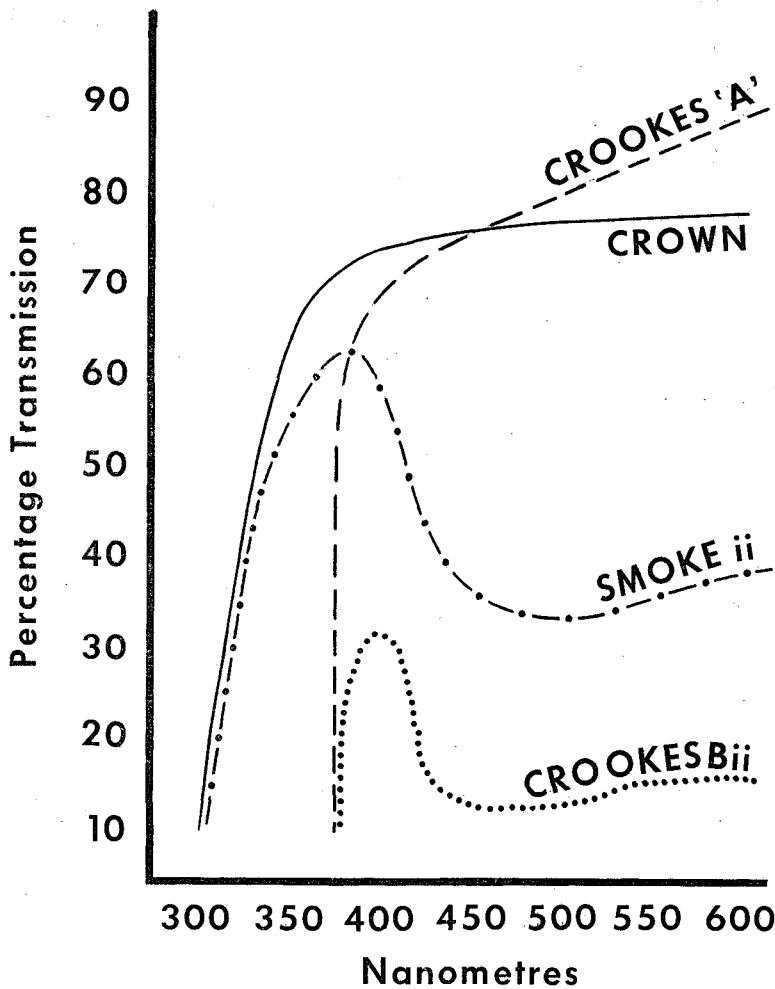


Fig. 1. Demonstrating the comparative effectiveness of different glasses in filtering U-VL of damaging wavelength.

thus producing specific DNA damage anti-bodies which might exacerbate the effects, on the corneas, of subsequent exposure.

Fig. 1, which is based on the work of Sir William Crookes (1914), demonstrates how easily U-VL of damaging wavelength can be filtered from sunlight and shows, inter alia, that ordinary Crown spectacle glass is, in itself, fairly effective. The graphs are for 2 mm thick glass. It will be seen that all Crookes' glasses effectively exclude U-VL of wavelength below 350 nanometres. Crookes glasses contain ferrous and cerium oxides.

When sunglasses are used to protect the corneal and conjunctival epithelia rather than the retinas (or the psyche) it should be remembered that subjective comfort does not imply effectiveness. Sunglasses will almost always prevent unscreened light from entering the pupils, but will not necessarily protect epithelia against light from above or reflected from below. Glasses should, therefore, be large and close-fitting.

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