Contamination of contact lens storage cases

Acanthamoeba is a free living protozoan that causes a rare but devastating corneal infection. The disease has emerged, principally among contact lens wearers in recent years. Although by far the largest number of cases have been reported from the USA,\(^1\) increasing numbers are also being seen in the UK.\(^2\) The disease is possibly rarer, under-diagnosed, or underreported in Europe.\(^3\)

The epidemiology is important because of the large numbers of lens users exposed to the risk of this potentially avoidable infection. It has been proposed that the recent increase in cases in the USA partly results from the growth in popularity of wearing contact lenses.\(^4\) Acanthamoeba is found in air, soil, and in salt, fresh, and chlorinated water and can be commonly isolated in nasopharyngeal cultures of human upper respiratory tract infections.\(^5\) Acanthamoeba keratitis has been shown in the USA to be associated with the use of home made saline solutions and swimming in contact lenses.\(^6\) As for bacterial keratitis in contact lens users,\(^7\) it has been shown that the solutions and containers for the care of lenses may be the source of the infecting organism. The majority of cases reported have been in users of soft lenses, though the disease is also found in association with the wearing of hard lenses.\(^8\)

Previous studies of asymptomatic wearers of contact lenses have shown Acanthamoeba present in home made saline but not in lens cases.\(^9\) Larkin et al, in their paper on the contamination of contact lens storage cases in this issue, have now shown that Acanthamoeba may be present in a high proportion of these in asymptomatic users of contact lenses. This difference may be related to improved culture success. Their finding parallels the situation for bacterial contamination of lens cases, which occurs in 35–52% of asymptomatic subjects.\(^10\) It has been expected because of the ubiquitous distribution of Acanthamoeba, the lack of effect of most cold lens disinfection systems against it,\(^11\) and the probability that the presence of bacteria enhances its survival and growth.\(^12\) Although the domestic water supply may be a source of this contamination, it remains only one of many potential sources of this widely distributed organism.\(^13\)

The link between contamination of lens care material and infection has established one of the risks for keratitis in contact lens users. Others are only now being established for bacterial keratitis. The factors influencing the adherence of organisms to a lens surface are complex\(^14\) and may or may not influence the spectrum of bacteria causing keratitis and their prevalence in users of different types of lenses.\(^15\) The relative risks of ulcerative keratitis in contact lens wearers have recently been shown to be up to 15 times higher for extended wear soft lenses than for daily wear soft lenses.\(^16\) It remains to be shown whether such considerations may also be important in Acanthamoeba infection.

These investigations have only just begun to unravel the pathogenesis and epidemiology of keratitis in contact lens users and many problems remain unsolved. The apparently higher incidence of amoebic keratitis in the USA might be explained by epidemiological comparisons with Europe, where lens use and hygiene practices, such as the use of home-made salines, may be different. The failure to link contaminated material for contact lens care with some cases of bacterial keratitis has not been explained.\(^17\) A recent case report on a user of disposable soft lenses has shown that this may also occur in Acanthamoeba keratitis.\(^18\) The reduced risk associated with hard lens wear, which Larkin et al have shown to be associated with a significantly increased rate of contamination of lens cases is also unexplained, and the risk of keratitis in rigid lens users has not yet been established. The problem of contamination by Acanthamoeba of material for the care of contact lenses is compounded by doubt about the efficacy of many cold disinfection systems against it.\(^19\) However, it is of equal concern that the same solutions meet rigorous standards of efficacy against bacteria in the laboratory but have been shown repeatedly to fail in use. Although this may be partly for reasons of poor compliance by the patient, it is possible that the failure may be due to the development of a bacterial biofilm that has been found on the surfaces of contaminated lenses in vitro.\(^20\) This is likely also to be important in container contamination and may have a role in resistance to disinfectants.

In order to create a safer environment for the contact lens user more needs to be known about both the epidemiology and the pathogenesis of lens related disease. This will be a continued challenge to the contact lens industry, regulatory bodies, and practitioners while contact lenses remain in widespread use.

JOHN DART

Albinism and anatomy

The average clinician going about his or her daily work may not have the time or the inclination to search for rational explanations for many phenomena the exact mechanisms of which are not apparent but which tend to be taken for granted. Examples of the sort of thing I mean would be ocular dominance, retinal rivalry, stereopsis, diplopia and confusion, and why convergent squints seem to cause amblyopia more often than divergent squints.

I have now discovered another riddle, and I am ashamed to have to admit that I did not know of its existence until I read the article in this month’s issue by Russell-Eggitt and colleagues. It appears that albinos have an anomalous chiasmal crossing arrangement, something I expect every ophthalmologist knows except me. Having heard of this anatomical oddity for the first time, I was prompted to wonder what lay behind it. At first sight it appears to be just an associated condition without any obvious causal relationship. This starts to seem unlikely, however, when we learn that all albinos have the anomaly; surely it must be very closely related to the albinotic state.

Digressing for a moment, may I remind readers of the discoveries of the last few years concerning the laying down of the visual pathways during postnatal development? The general principle which has been established is that the development of the intracerebral visual pathways depends to some extent on the input of appropriate stimuli. We have been told, for example, how the anatomy of the lateral geniculate body can vary according to whether or not occlusion has been applied to one eye in experimental animals, and the same sort of thing has been suggested in the occipital cortex.

The situation in albinism, as explained and illustrated in the current article, is that in the central 20° of the field in one eye, retina stimulated by light coming from the opposite side, which would normally be expected to project to the ipsilateral cortex, does not do so but projects back to the opposite side. (I prefer to talk about the 'opposite side' rather than the 'nasal side' because it has more meaning in the context of what comes next.) I found this revelation profoundly disturbing for the following reason. I have always thought (and taught) that everything we 'relate to' (to use a bit of slightly unpalatable but I hope in this case appropriate jargon) on one side of our world is processed on the other side of our brain. This shows itself in many ways, not least in a variety of forms when hemicranial dysfunction occurs for one reason or another. The simplest example is in the unawareness of loss in many patients with homonymous hemianopia. It is not that they cannot see one side, but that one side from a visual point of view has ceased to exist; they cannot even think about it.

This strict concept of 'side' implies a changeover point where right gives way to left at the centre. There is probably a small area at the centre which the brain recognises as centre and where the concept of one or other side does not operate. Such is probably true of the most basic of bodily functions, eating, excreting, and reproducing. We can probably add central vision as the fourth 'centralised' function. It is possible that, whereas the right and left sides are mediated by the opposite sides of the brain, central functions are bilaterally represented (sparring of the macula might be an example of this). Perhaps therefore the peculiar chiasmal arrangements of the albino are not so difficult to understand after all. Because their central vision is poor from an early age they may have developed a much less concentrated version of the 'centre'. Putting it another way, we may say they may have an extended central zone where right and left are not perceived as such but are dissolved into a large (20°) blurred centre. If this were to be the case I should expect a wide central area to be bilaterally represented in the cortex, and this might account for the anomalous decussation found in albinos. It would be interesting to have the views of an expert on this speculative explanation.

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