Editorial: The case against the lens

It is well known that after a perforating injury to the lens cataractous changes and inflammatory reactions follow in the traumatised eye. A similar inflammatory reaction may develop in the other eye at a later date, perhaps more violently if it harbours a cataract or is injured. In the past, especially before the steroid era, when extracapsular lens extraction was a common procedure, the incidence of lens-induced uveitis was high. The involvement of the unoperated eye after this procedure was first noted by Straub (1919), and this phenomenon was later well documented by, among others, Courtney (1942), Irvine (1957), Witmer (1957), and Wirostko and Spalter (1967); in some cases, however, the superimposition of sympathetic ophthalmitis could not be ruled out. It is of interest that Easom and Zimmerman (1964), in a report of 7 cases, noted that whereas the exciting eye showed the picture of sympathetic ophthalmitis the sympathising eye had lesions of both sympathetic ophthalmitis and phacoanaphylaxis in 6 cases; in further 4 cases in which the exciting eye could not be examined the so-called sympathising eye showed only phacoanaphylaxis histologically. Blodi (1959) believes that hypersensitivity to lens proteins may play some part in the causation of sympathetic ophthalmitis.

Since planned extracapsular extraction is rarely undertaken in the Western world, the incidence of postoperative lens-induced uveitis has declined sharply, but it is still a daunting problem in the developing world, where not only is the incidence of cataract high but it also occurs at a much earlier age, and owing to socioeconomic reasons and manpower problems a large number of lenses are removed by the extracapsular method. There has been renewed interest in this problem because of the increased incidence of ocular injury due to road traffic accidents. A failure by ophthalmologists to treat such cases more drastically and to remove the injured lens at the time of wound repair may lead to clinically unsuspected phacoanaphylaxis (Perlman and Albert, 1977). If the other eye is injured or operated on at a later date, a violent lens-induced inflammation may develop.

In the past 3 distinct types of lens-induced ocular inflammation were recognised. The first was a severe form called endophthalmitis phacoanaphylactica or phacogenic uveitis; it showed an initial polymorphonuclear infiltration which later developed a granulomatous reaction characterised by palisading mononuclear phagocytes, epithelioid cells, and giant cells. The second was a less severe form called ‘phacotoxic uveitis’, which showed features of a mainly non-granulomatous inflammation. The third was phacoletic glaucoma, in which liquefied lens protein from a hypermature cataract attracted a large number of phagocytic mononuclear cells which blocked the filtration angle, leading to a raised intraocular pressure. Phacoletic glaucoma is now considered to be a non-immune phenomenon and there is no evidence that lens protein is toxic. Since anaphylaxis is now used to signify a special class of allergic reaction mediated by IgE, the term endophthalmitis phacoanaphylactica has a limited use. Rahi et al. (1977) have suggested that the condition be called phacoallergic endophthalmitis, in which the inflammatory process involves not only the uvea but also the lens and the vitreous and which can be produced by both antibody as well as T-cell mediated hypersensitivity reactions (Marak et al., 1976; Kincses and Szabo 1976).

It should be realised, however, that the evidence that autologous lens proteins are involved in ocular allergic reactions has so far been indirect. Since the first demonstration that heterologous lens proteins are antigenic (Uhlenhuth, 1903) experiments have been carried out to establish that an immune reaction to lens proteins may lead to ocular inflammation. These studies have been carried out, however, with either xenogeneic (Verhoeff and Lemoine, 1922), allogeneic (Goodner, 1964) or syngeneic (Behrens and Manski, 1973) material, and although antilens antibodies have been detected in lens-induced uveitis in man (Wirostko and Spalter, 1967) there has been no evidence so far that autologous lens proteins are antigenic. In this issue of the journal Rahi, Misra, and Morgan (p. 371) show for the first time that not only are autologous lens proteins antigenic but they stimulate both B- and T-lymphocyte activity. The allergic response in their animals was variable, however, suggesting a modulatory role played by the immune response genes which are believed to be linked to the major histocompatibility system. Thus every ocular injury does not lead to an autoimmune inflammation; in fact only about a quarter of the patients develop uveal inflammation of any degree after unplanned extracapsular extraction.

Since it is now known that soluble lens proteins may be present in small amounts even in the aqueous humour of individuals with normal lenses (Sandberg, 1976), it would be interesting and
valuable to examine preoperative blood specimens from patients with cataracts for evidence of lens haemagglutinating antibody and T-cell sensitisation—these immunological tests according to Rahi et al. (1977) appear to be the most reliable indicators of lens allergy—and to see if the individuals with positive tests are more likely to develop post-operative complications. This information would be essential in anticipation of an uncomplicated lensectomy.

References


