

Editorial: Postmeasles blindness

In Africa measles is a major cause of corneal blindness, which is known as postmeasles blindness (PMB). Various surveys indicate that measles is responsible for 14 to 81% of childhood blindness. In a prospective study of measles in a Kenyan mission hospital keratitis was observed in most children with measles (76%).¹ In most cases this keratitis healed without treatment and without sequelae. However, a small proportion of children (<5%) later developed corneal complications consisting of large corneal erosions or exposure ulcers, leading to secondary infection, corneal ulcers, and perforation. In another study herpes simplex virus was isolated from almost half and measles virus from over 10% of children with active corneal ulcers following measles.²

The factors which lead to the development of PMB are not well defined. It has been suggested that malnourishment, particularly vitamin A deficiency, associated immunosuppression, and genetic factors are all involved in the development of PMB. Studies have shown that there did not appear to be any correlation between the extent and severity of measles keratitis and nutritional status.¹ In addition there were no differences in serum prealbumin levels or serum retinol-binding protein levels between malnourished children with corneal ulcers after measles compared with other malnourished children without corneal disease after measles.²

The role of immunosuppression in the development of PMB is not clear. PMB is not seen in immunodeficient children with leukaemia in developed countries, who fail to develop a measles rash. While malnourished children may be partially immunosuppressed, they develop immune responses to measles virus during infection. They produce normal levels of antibodies and a severe measles rash, which is the result of cell-mediated immune responses to measles virus. However, these children also shed measles virus for longer, and their sera have inhibitory activity in some in-vitro tests of cell-mediated immunity.³ It has been suggested that genetic factors contribute to the development of PMB. In African children measles is particularly severe and PMB is common, whereas in Asian children PMB is not common. It may be that African children are particularly susceptible to severe measles and PMB.

The paper by Kogbe and Liotet which appears in this issue describes their studies of tears from children with severe measles. They found that children with measles were deficient in tears. This may suggest that measles virus interferes with normal tear production in some way. This lack of tears must render the eyes of these children particularly vulnerable to secondary infections. They also found that in the early stages of measles children had a normal tear protein pattern or a 'serum' protein pattern due to the transudation of serum proteins through blood capillary walls during acute inflammation. In contrast malnourished children with corneal erosions in the later stages of measles had considerable amounts of transferrin but no other serum proteins in their tears. However, the presence of transferrin in tears was not due to malnourishment, since malnourished children without measles had normal tear protein patterns.

These results raise several important questions. Does measles always cause a marked reduction in tears or are malnourished African children particularly susceptible? Does this deficiency in tear production contribute to the development of PMB? Why do large amounts of transferrin appear in tears, and are the physical properties of tears altered? Does transferrin contribute to the development of PMB, in which case does it appear before, during, or after the development of PMB, or is it a marker of corneal erosions? Measles and PMB can be prevented by vaccination, but until vaccination is universally implemented PMB will continue to cause preventable blindness. In the meantime a better understanding of the factors involved in the development of PMB could lead to the development of cheap, simple, and effective methods of treatment and prevention, which are urgently needed.

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References

- 1 Dekkers NWHM. The cornea in measles. In: Darrell RW, ed. *Viral diseases of the eye*. Philadelphia: Lea and Febiger, 1985: 239-50.
- 2 Sandford-Smith JH, Whittle HC. Corneal ulceration following measles in Nigerian children. *Br J Ophthalmol* 1979; **63**: 720-4.
- 3 Whittle HC, Mee J, Werblinska J, Yakubu A, Onuora C, Gomwalk N. Immunity to measles in malnourished children. *Clin Exp Immunol* 1980; **42**: 144-51.