Childhood blindness from corneal ulceration in Africa: causes, prevention, and treatment*

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Corneal scarring is responsible for approximately 70% of blindness in children in African countries. Recent studies confirm that measles infection is a common predisposing factor and that vitamin A deficiency is responsible for the majority of bilateral corneal ulceration. Herpes simplex keratitis (caused by human (alpha) herpesvirus types 1 and 2), ophthalmia neonatorum, and the use of traditional eye medicines also constitute significant causes of corneal ulceration in children in Africa. Intervention strategies, the management of corneal ulceration in children, and areas for future investigation are discussed.

Surveys of blind children in Malawi (1), the United Republic of Tanzania, Ethiopia (A. Foster, unpublished observation), and Nigeria (2) have shown that approximately 70% of the blindness resulted from corneal scarring. In India, Indonesia, and other parts of south-east Asia, vitamin A deficiency is a well-established cause of corneal scarring and blindness in children (3). However, concerning the etiology of corneal scarring in children in Africa there is controversy on the relative importance of vitamin A deficiency (xerophthalmia) and other factors, such as keratitis caused by human (alpha) herpesvirus 1 or 2 (formerly called herpes simplex virus types 1 and 2), the use of traditional eye medicines, measles infection, and exposure keratopathy (2, 4–6).

It is very difficult to identify the cause of corneal scarring in children several years after the corneal ulceration. Many blind children show evidence of previous large and deep

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corneal ulceration suggestive of vitamin A deficiency, but the same type of clinical appearance can be seen after the use of traditional eye medicines. Other blind children have small shrunken eyes (phthisis bulbii) from which it is usually impossible to determine the nature of the original eye disease. Thus, although corneal ulceration, leading to corneal scarring, is responsible for almost three-quarters of childhood blindness in Africa, the cause of the ulceration often cannot be determined.

PREDISPOSING FACTORS

Measles

Sauter’s investigation of blind schoolchildren in the United Republic of Tanzania in 1978 showed that about 50% had a history of recent measles infection before going blind, a similar figure (44%) was attributed to post-measles blindness among Malawian blind schoolchildren (1).

Measles virus often invades the corneal epithelium causing a superficial punctate keratitis, which usually resolves as the measles rash clears (5). Three hospital studies in the United Republic of Tanzania (at Kilimanjaro Christian Medical Centre, Mvumi Hospital and Tembeke Hospital) showed that true corneal ulceration, as distinct from superficial punctate keratitis, followed acute measles infection with an attack rate of 2.5%, 3.9% and 4.0%, respectively, which is similar to findings elsewhere (7). Surveillance of corneal ulceration in children carried out by trained eye auxiliaries over a two-year period in the same country showed that half of the unilateral corneal ulcers, and more than three-quarters of bilateral ulcers occurred within three months of measles infection (8); and in a hospital study the present authors found that over half of 49 bilateral ulcers in children occurred within one month of measles infection (9).

There can be little doubt that measles infection is the precipitating factor for at least half, and maybe three-quarters, of all bilateral corneal ulcers and resulting blindness due to corneal scarring in childhood; and that 1-4% of African children with acute measles will develop corneal ulceration. Furthermore, it has been observed that children with chronic diarrhoea and malnutrition, following measles, are at risk of developing corneal ulceration many weeks or even months after the acute measles infection.

Xerophthalmia

Vitamin A deficiency presents in its early stages as night blindness, and then dryness of the conjunctiva with Bitot’s spot formation. Later, there is dryness of the cornea and then frank ulceration of the cornea. Although the occurrence of xerophthalmia has been reported in Kenya (6, 12), the United Republic of Tanzania (10), the Luapula Valley in Zambia (11), Matabeleland in Zimbabwe (13) and Nigeria (14, 15), its importance has also been questioned (2, 4, 5).

The two-year surveillance in the United Republic of Tanzania revealed cases of night blindness and Bitot’s spot formation that were unrelated to measles infection in all the 11 regions studied. Prevalence rates could not be established, however, because the children examined were a selected group of hospital referrals (8).

In a study of 5436 under-six-year-old children in the Lower Shire Valley, Malawi, the prevalence of xerophthalmia was found to be: conjunctival xerosis (X1A) 0.9/1000, Bitot’s spots (X1B) 3.3/1000, and corneal xerosis or ulcer (X2/3) 0.6/1000. Xerophthalmia was responsible for 42% of corneal scars in preschool children, with a prevalence rate of xerophthalmic corneal scarring (XS) of 5.9/1000 (16).
Vitamin A deficiency accounted for 84% of bilateral corneal ulcers in children in the Tanzanian surveillance study, and 55% of bilateral corneal ulcers in one Tanzanian hospital (9). In the Malawi survey all six cases of bilateral blindness in preschool children were attributed to xerophthalmia, and in the Tanzanian hospital study 69% of the children deemed to go blind from bilateral corneal ulceration had clinical xerophthalmia. There are therefore good data from community-based prevalence studies and clinical investigations in these two countries to confirm the presence of early xerophthalmia in preschool children and the importance of vitamin A deficiency in the etiology of bilateral corneal ulceration and childhood blindness.

Other causes

Herpes simplex keratitis (or keratitis caused by human (alpha) herpesviruses), trauma, and bacterial infection were all reported to cause unilateral corneal ulceration in the Tanzanian surveillance study which documented 294 cases of corneal ulceration in children, of which nearly two-thirds were unilateral (8). In a three-year prospective study at one hospital in the United Republic of Tanzania, herpes simplex keratitis was the commonest cause of unilateral corneal ulceration, often presenting as an amoeboid type of ulcer in association with measles, malaria or other pyrexial illness (9). Human (alpha) herpesviruses were isolated from 47% of 34 corneal ulcers in Nigerian children (2), where they were thought to be a major cause of corneal ulceration. Herpes simplex ulceration is usually unilateral and superficial and therefore does not commonly lead to blindness.

Other causes of bilateral ulceration are the use of harmful traditional eye medicines and ophthalmia neonatorum, both of which were responsible for a significant number of cases in the Tanzanian hospital study. Traditional eye medicines are usually herbal in origin and sometimes dissolved in animal or human urine. They are applied on top of some pre-existing eye disease which may be measles, conjunctivitis or corneal ulceration. The use of traditional eye medicines and their destructive capacity varies widely from place to place; however, the ulceration is often bilateral and severe, leading to blindness.

PREVENTION STRATEGIES AND TREATMENT

Accepted that corneal scarring is the commonest cause of blindness in children, and that vitamin A deficiency is present in preschool children and is the most important cause of bilateral corneal ulceration, an effort should be made to improve the vitamin A intake of young children. There are three possible prevention strategies.

— Firstly, to improve the diet of preschool children through a nutrition programme which stresses the importance of readily available foods rich in vitamin A, e.g., dark green leafy vegetables and papaya; this should be part of the general education of mothers and children on the subject of good nutrition.

— Secondly, regular distribution of vitamin A capsules containing 110 mg retinol palmitate (200 000 IU, every six months) either to all preschool children, or to those children at high risk of developing xerophthalmia, i.e., children with marasmus, kwashiorkor, early signs of vitamin A deficiency or severe measles.

— Thirdly, the fortification with vitamin A of a food which is consumed by preschool children and which is generally available and centrally processed — sugar would appear to be the most suitable — although the cost of such a programme would be considerable.
All these programmes will present problems in implementation, though ideally the nutrition education programme will have the longest-lasting and most cost-effective impact. A compromise policy would be the distribution of vitamin A capsules to those children at high risk, as a short-term strategy, combined with national programmes for education in good nutrition as a long-term objective.

Considering that 50-75% of the cases of childhood corneal ulceration and subsequent blindness are associated with measles, immunization against this disease would do much to reduce not only childhood mortality, but also childhood blindness. Herd immunity can be expected when immunization cover reaches 80%, but this figure is rarely attained in many African countries. The expanded programmes on immunization, however, may enable measles immunization coverage rates to increase in the near future, with a consequent reduction in childhood mortality and morbidity from blindness.

Management of corneal ulceration in children

The following prevention and treatment schedules are suggested for areas of Africa where corneal ulceration and corneal scarring in children occur, or where protein-energy malnutrition is a problem.

Prophylactic administration of vitamin A, 200 000 IU orally, as a single dose should be given to children with:

— marasmus or kwashiorkor;
— severe measles;
— night blindness, conjunctival xerosis or Bitot’s spots.

This will protect children with severe malnutrition and severe measles from vitamin A deficiency, and will treat those children with early manifestations of xerophthalmia.

If a child is seen with a suspected corneal ulcer, then the following treatment is recommended:

1. Immediate administration of vitamin A, 200 000 IU orally (or injection of aqueous vitamin A, 100 000 IU, if the child is vomiting); repeat after one day and again after one week.
2. Antibiotic eye ointment (e.g., 1% tetracycline) three times per day.
3. 0.5% atropine eye ointment, once daily.
4. An eye pad.
5. General treatment for malnutrition, diarrhoea and other systemic infections.

If the corneal ulcer fails to improve within five days on this regimen, it may indicate infection with human (alpha) herpesvirus. This will require additional treatment with antivirals (e.g., idoxuridine or trifluridine), or possibly mechanical debridement of the corneal epithelium if the ulcer is still superficial.

It is very important for all health workers to be on the lookout for the early development of corneal ulceration in any child with malnutrition or measles; failure by the child to open its eyes is often indicative of corneal ulceration in these children.

RESEARCH NEEDS

Recent findings in Africa have confirmed the importance of vitamin A deficiency and measles as a cause of childhood blindness, but they have also shown that other factors are involved in both measles-associated corneal ulceration and in non-measles ulceration in children. Some of the questions that remain unanswered include:
— Why is herpes simplex (human (alpha) herpesvirus) infection so common, especially in one- and two-year-old children, and is it related to their nutritional status?
— How do measles (and malaria) precipitate herpes simplex ulceration?
— Is fever the trigger or is the depression of cell-mediated immunity important?
— How widespread is the use of traditional eye medicines, what do they contain, and how can their use be controlled?

It is hoped that the next few years will see a reduction in childhood blindness as programmes for measles immunization and vitamin A supplementation come into effect, and that further research will help in the better understanding of the mechanisms involved in corneal ulceration from human (alpha) herpes viral infection and the use of traditional eye medicines.

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