

Childhood blindness in Tanzania

THIS paper was prepared under Cooperative Agreement No. AID/DSAN-CA-0267-931-0045 between the International Centre for Epidemiologic and Preventive Ophthalmology, John Hopkins University and the Office of Nutrition, United States Agency for International Development. Those involved in the project included Dr. Fatma Mrisho, Dr. Kinabo, Dr. Frits Van der Haar, Dr. Malentema, Prof. V. Kimati, Dr. John McKigney and Dr. Alfred Sommer of the International Centre for Epidemiologic and Preventive Ophthalmology of Wilmer Ophthalmological Institute at the John Hopkins School of Hygiene and Public Health who wrote the final report. The International Centre for Epidemiologic and Preventive Ophthalmology is a World Health Organisation centre for the prevention of blindness.

IN MANY areas of Africa, including Tanzania, measles is considered the principal cause of childhood blindness. Corneal ulceration and necrosis accompanies measles in Kenya², Nigeria³, Zambia⁴, and other countries. Chirambo and colleagues demonstrated that a majority of children in schools for the blind claimed their problem began with measles⁵, a fact confirmed during a recent visit by Sommer⁶. The size of the problem, and its cause, remain unknown.

MEASLES BLINDNESS IN TANZANIA

There is little doubt measles blindness occurs in at least some areas of Tanzania (1). Sauter reported numerous cases in children hospitalised on the measles ward at the Muhimbili Medical Centre⁷. Professor Kimati and Dr. Sharma confirmed these observations, and Dr. Mmbaga has seen similar cases in Dodoma. Fewer cases are reported from Mwanza and Moshi.

J. Manyanga examined the eyes of all children admitted to the measles ward at Muhimbili between June and July, 1977⁸. Roughly 14 per cent were thought to have some form of corneal involvement, and 2 per cent corneal destructive lesions. A majority of children admitted to the eye ward at Muhimbili with corneal destruction associated their disease with an episode of measles (9).

CAUSE OF MEASLES BLINDNESS

During measles infection, the virus replicates in the corneal epithelium, producing red, inflamed eyes, and causing many children to keep their eyes closed. But otherwise healthy and well children in developed countries do not develop corneal ulceration and necrosis. What then is responsible for the severe ocular in African children? Several factors have been suggested.

• Vitamin A deficiency: One of the strongest candidates is vitamin A deficiency. Severe vitamin A deficiency results in corneal ulceration and necrosis identical to that observed in measles¹⁰. Measles interferes with vitamin A metabolism, and in Indonesia children, precipitates half the cases of blinding xerophthalmia¹¹.

Some Indonesian children presenting with severe measles already had corneal ulceration. Others developed corneal destruction 2-4 weeks later, in association with a deterioration in their general nutritional status. All had severely depressed vitamin A levels. Most were also protein deficient, which also impairs vitamin A metabolism.

The picture amongst Tanzanian children appears to be similar. In a brief visit to the pediatric wards at Muhimbili, Mrisho and Sommer did not find any cases, that day, on the measles ward, but found 4 cases of active corneal destruction among malnourished children on the general wards who suffered a measles attack 2-4 weeks earlier.

Further, circumstantial evidence for the role of vitamin A deficiency comes from reports by Dr. Kinabo and others that blindness is much less frequent among measles cases in Mwanza, where fish (whose livers are a good source of vitamin A) are widely



IN many parts of Africa, including Tanzania, measles is considered the principal cause of childhood blindness.

consumed. It should also be remembered that corneal destruction commonly accompanied measles in 18th and 19th century Europe and America, when the general nutritional status was poorer.

Some workers have observed measles blindness is uncommon in African populations that consume Red Palm Oil, a potent source of provitamin A carotenoids.

Although evidence in support of malnutrition is strong, it is not conclusive. If measles were merely precipitating xerophthalmia in vitamin A and protein deficient children one would expect to encounter some children with mild xerophthalmia (night blindness and Bitot's spots), and see some patients with typical corneal destruction without a recent history of measles. Both Professor Kimati and Dr. Sharma have seen a few such children but they are particularly rare.

It is very possible, however, these children have not simply been presented to hospitals. In a recent visit to the Lower Shire Valley in Malawi, Sommer observed children with typical corneal ulceration and kwashiorkor. Two had recently had measles but the third allegedly had not.

• Other causes: other causes suggested include application of traditional drugs to eyes of measles children resulting in secondary bacterial or chemical keratitis; immuno-suppression and secondary herpes keratitis; and unknown factors.

RECOMMENDATIONS

In February 1981, a first national seminar on the problem of vitamin A deficiency in Tanzania was held at the Institute of Finance Management, Dar es Salaam. It drew participants from the Tanzania Food and Nutrition Centre, Muhimbili Medical Centre, Regional Ophthalmologists (AMO's) from Dodoma, Arusha and Iringa and representatives from the Ministries of Health and Agriculture. The seminar discussed the problems of vitamin A nutrition, its relation to various diseases in particular measles, and made recommendations on launching vitamin A deficiency control measures.

Given the serious nature of the problem, the Vitamin A Co-ordinating Committee decided it requires urgent investigation.

Two lines of inquiry were suggested: try and determine whether vitamin A and protein deficiency, or other factors were responsible for measles blindness; and attempt to document the presence or absence of *xerophthalmia* in Tanzania.

•Measles Blindness: It was proposed that all children admitted to Muhimbili have a careful eye examination, and be followed regularly for up to one month. Those who develop typical corneal ulceration and matched controls, would be intensively studied. Parameters of concern include vitamin A and protein status; general systemic status (malaria, tuberculosis); severity of measles; presence of herpes virus; etc.

The results would be compared with children of similar age, sex, and measles severity but without corneal ulcers. Similar though simpler studies on measles cases in Mwanza would confirm or disprove the belief that regional variations exist. Results would be further confirmed by a therapeutic/diagnostic trial of the use of vitamin A.

Children with corneal ulcers, and age/sex controls with normal corneas admitted to the general pediatric wards at Muhimbili will be studied in similar fashion, since many of the blinding cases present after the acute measles episode have subsided.

•*Xerophthalmia*: Given the strong suggestions that vitamin A deficiency is at the heart of the problem, it was decided to undertake a relatively simple search for pure *xerophthalmia* before deciding to launch a detailed survey. All AMO-Ophthalmologists would attend a short training seminar in the recognition of *xerophthalmia*. For one year, they would examine, once a week, all children hospitalised with malnutrition, keeping a simple record of the numbers examined.

All children found to have *xerophthalmia*, of whatever severity, would have their age, sex, clinical changes and measles history recorded. The absence of *xerophthalmia* cases in a particular hospital does not prove *xerophthalmia* is absent from that region, since cases might simply not come to the hospital. But identification of large numbers of cases would suggest that a serious problem of vitamin A deficiency exists in the region, requiring further investigation and attention.

GOALS

The major results of both studies should provide sufficient

information to answer two basic questions, both for Tanzania and Africa as a whole: is vitamin A deficiency the mechanism by which measles results in blindness, and is *xerophthalmia* an important problem in its own right. The sooner these studies get underway the better; and the sooner the questions are answered, the fewer the number of children who will continue to be blinded each year.

REFERENCES:

1. Franken, S: Measles and *xerophthalmia* in East Africa. Trop. Geogr. Med. 26: 39-44, 1974.
2. Sauter, J: *Xerophthalmia* and measles in Kenya. Drukkerij Van Dendren, B. V. Groningen. 1-235, 1976.
3. Oomen, J. M. V: *Xerophthalmia* in Northern Nigeria Trop. Geogr. Med. 23: 246-249, 1971.
4. Awdry, P. N., Cobb., Adams, P. C. G: Blindness in the Luapula Valley. Cent. Afr. J. Med. 13: 197-201, 1967.
5. Chirambo, M. C., BenEzra, D: Causes of blindness among students in blind school institutions in a developing country. Br. J. Ophthalmol. 60: 665-668, 1976.
6. Sommer, A: Brief Report on Measles and *Xerophthalmia* in Malawi. March, 1981. ICEPO, Johns Hopkins Hospital.
7. Sauter, J. J. M: Measles in Tanzania. Report to the First General Assembly, International Agency for Prevention of Blindness, Oxford, 1978.
8. Manyanga, J. S. N.: Ocular involvement in relation to general complications in severe measles. Elective Period Research Project, 1977.
9. Kinabo, N. N.: Keratomalacia in children as seen at the eye-ward of the Muhimbili Medical Centre. Read at the Annual Meeting, Medical Association of Tanzania, 1979.
10. Sommer, A., Sugana, T: Corneal *Xerophthalmia* and Keratomalacia. Arch. Ophthalmol. (in press).
11. Sommer, A: Nutritional Blindness: *Xerophthalmia* and Keratomalacia. New York and Oxford, Oxford Press, 1982 (in press).
12. Vitamin A Deficiency in Tanzania: Report of a National Seminar. TFMC, Medical Nutrition Department, Report No. 650 (1981).