Original article

Xerophthalmia at a welfare home in Jimma town

Negussie Zerihun[1]

Abstract: One hundred and seven children under sixteen years of age were examined between May 1994 and Octobr 1995; of whom 39 (36.5%) had evidence of clinical xerophthalmia. Twenty three (58.97%) of these xerophthalmic children were males (M:F=1.4:1). Thirty six children (92.3%) were over seven years of age. No significant association was seen between gender and xerophthalmia ($p=0.94$). Respiratory tract infections and diarrhoea were seen in 30.8% of xerophthalmic and 35.3% of non-xerophthalmic children, respectively, with no significant difference in morbidity pattern between the two groups ($p>0.5$). Seven of the xerophthalmic children were wasted while two were stunted. Chronic malnutrition (stunting) was significantly associated with xerophthalmia ($p<0.05$). Night blindness and bitot's spots disappeared within three weeks of initial vitamin A administration. Bitot's spots couldn't vanish completely in two children. It is recommended that the welfare home administration has to provide the children with cheap and locally available vegetables that are rich in vitamin A; and in the long run become self-sufficient by developing its own garden. [*Ethiop. J. Health Dev.* 1997;11(3):213-218]

Introduction

Vitamin A deficiency (VAD) is the main cause of preventable childhood blindness (1,2). Its severe form affects up to 500,000 young children every year; most of whom are in the developing world (3). Although the deficiency state can affect any age group, the most susceptible are preschool children(4). Displaced children from natural calamities, may be at a greater risk of VAD (5).
The same may apply to institutionalised children if their diet is deficient in the nutrient.

VAD is known to be an important public health problem in Ethiopia (6-8). Nutritional education of the public has been advocated as an important preventive tool against childhood blindness. The impact of dark-green leafy vegetables(dglv) and fruits in the prevention of vitamin A deficiency has been a universally accepted knowledge until recently. Some works have now imposed serious challenges by questioning the efficacy of plant sources of provitamin A to improve vitamin A status (9,10).

The impact of vitamin A on childhood morbidity has been another controversial issue. The association between vitamin A deficiency and increased childhood morbidity is well documented(11-14). Hence, it was generally agreed that supplementation of the vitamin would reduce childhood morbidity from diseases like diarrhoea and respiratory tract infections. But reports have appeared to demonstrate that supplementation has little lowering effect on childhood morbidity(15-18).

The use of vegetables and fruits in the prevention of xerophthalmia will, however, continue specially in developing countries where retinol-rich foods are difficult to come by.

The present study was initiated by an encounter, during a routine out-patient activity at Jimma Eye Unit (JEU), of three children with mild xerophthalmia on a single morning. All of them were from a near-by welfare home. With the belief that more cases of VAD may be available at the home, the study had been undertaken with the following objectives:

[1] From the Department of Ophthalmology, Jimma Institute of Health Sciences, P.O. Box 378, Jimma, Ethiopia.

a. to study the prevalence of VAD in children under sixteen years of age who are residents of the

home.

b. to compare morbidity patterns in the previous one month between xerophthalmic and nonxerophthalmic children
c. to study the regression pattern of the signs and symptoms of VAD
d. to come up with a feasible long-term solution to the problem of VAD at the home.

Methods

There were a total of one hundred and seven children under sixteen years of age at the home all of whom were taken for the study. The names, ages, gender, weights, morbidity pattern during the previous month, history of eye trauma, eye infection or application of traditional eye medications were inquired and responses obtained from older children and/or foster mothers recorded. Diarrhoea was defined as the passage of liquid stools at least three times a day. Respiratory infection was diagnosed if the child had cough with fever for at least three days. Urinary infections were documented if there was a history of dysuria or frequent urination with fever and/or chills; while skin infection implied any dermatological condition, including acne-like lesions, scabies or boils.

Weights and heights (lengths) were each measured twice to the nearest 100 grams and 0.5 cms, respectively, by two different recorders trained for the task. The average measurement was taken for each child.

Night blindness, which has an Amharic equivalent of "dafint", is well known in the area. It was thoroughly explained to older children and foster mothers. Older children were asked if they find it difficult to walk about, or play, at dusk; and if younger children stumble over objects at conditions of reduced illumination. The presence or absence of night blindness was documented only after its meaning was well understood. When any uncertainty was noted, the case was automatically recorded as negative (i.e no night blindness). Visual acuity (v/a) was recorded in cooperative children over four years of age using the Snellen E-chart.

Examination of the anterior segment of the eye was done using a torch and a magnifying loupe (2x magnification). Children suspected of clinical VAD were subjected to fluorecein staining of the cornea for subsequent slit lamp examination at JEU. Direct ophthalmoscopy was done in xerophthalmic children after the application of two drops of 1% Cyclopentolate drops into each eye.

Nutritional status was evaluated according to weight-for-height and height-for-age, standard deviation scores or z-scores using the Anthro Version 1.01 anthropometry Soft Ware (Nutrition, CDC and WHO; Dec., 1990). Children with ZWH score of <=-2 were considered wasted and those with ZHA score of <=-2 were considered stunted.

Clinical xerophthalmia was diagnosed if a child had a history of night blindness(XN), or when ocular examination revealed Bitot's spots (XIB), or corneal xerosis (X2). Conjunctival xerosis (X1A) occurring together with night blindness was also considered diagnostic of clinical xerophthalmia. Serum retinol determination was not done.

Xerophthalmic children were given three doses of vitamin A (retinol palmitate 200,000 IU) on days one, two and seven. They were then put on prophylactic doses of vitamin A (200,000 IU) every six months for the next eighteen months. The rest of the study population got a single dose followed by a six monthly administration. Children under one year of age received half the dose. Response to therapy in terms of amelioration of night blindness and disappearance of Bitot's spots was checked weekly for the first two months, fortnightly for the next four months and monthly for the remaining twelve months. Statistical analysis was carried out using Epi-info version 6.

Results

Of the total one hundred and seven children examined, sixty four were males and forty three females (Table 1). Thirty nine children (36.5%) had clinical xerophthalmia, of whom only three were seven years of age or under whereas the majority (92.3%) were older than seven years (Table

2). Twenty two other children had conjunctival xerosis only and three others had corneal scarring that could not be attributed to VAD alone.

Table 1: **Age and sex distribution of children at the Jimma welfare home, 1995**

Age(years)	Male		Female		Total
	Xeroph.	Non-xeroph	Xeroph	Non-xeroph	
Under 4	-	6	1	4	11
4 to 7	1	7	1	9	18
8 to 11	15	8	7	7	37
12 to 15	7	20	7	7	41
Total	23	41	16	27	107

Twenty three of the xerophthalmic children were males (58.97%) and sixteen were females (41.03%) with a male to female ratio of 1.4:1. No statistically significant association was seen between xerophthalmia and gender (p=0.94). Ninety children had visual acuity of $\geq$ 6/18 in the better eye. V/a couldn't be determined in 17 children who were either uncooperative or too young.

Table 2: **Signs and symptom of VAD in children at the Jimma walfare home, 1995**

Age (years)	XN	XN+XIA	XIB	X2	Total
0 - 3	-	-	1	-	1
4 - 7	-	-	2	-	2
8 - 11	1	2	19	1	23
12 - 15	2	1	10	-	13
Total	3	3	32	1	39

Respiratory tract infections (RTI) and/or diarrhoea were responsible causes of morbidity in 30.8% of xerophthalmic and 35.3% of non-xerophthalmic children. No statistically significant difference was seen in morbidity pattern between the two study groups (p>0.5) (Table 3).

Seven of the 39 (17.9%) xerophthalmic and fourteen of the 68 (20.6%) non-xerophthalmic children had wasting while six (15.4%) of the xerophthalmic and two (2.9%) of the nonxerophthalmic children were stunted. One xerophthalmic child had both wasting and stunting. Xerophthalmia was associated with chronic malnutrition (P<0.05). No significant difference was seen in the prevalence of acute malnutrition in the two groups of children (p=0.94) (Table 4).

Table 3: **Morbidity pattern among the study populations, Jimma, 1995**

Causes of Morbidity	Xerophth Children		Non-xeroph Children			
	No	%	No	%	X^{2*}	P-value
R.T.I &/or diarrhoea	12	30.8	24	35.3	0.23	0.23
Skin infections	2	5.1	1	.5	1.21	1.21
U.T.Infections	1	2.6	2	2.9	0.01	0.01
None	24	61.5	41	60.3	0.02	0.02
Total	39	100	68	100		

* Mantel-Haenszel test

** Fisher exact 2-tailed values

Night blindness disappeared within a week of initial therapy. Ten children (25.6%) and twelve others (30.8%) with Bitot's spots showed complete disappearance of the lesions within two and three weeks, respectively, of initial treatment. Two children had marked shrinkage of Bitot's spots that failed to go away within eighteen months (Table 5). Two children in the xerophthalmic group (who had responded completely) and one child in the non-xerophthalmic group developed Bitot's spots about 56 weeks after the beginning of the study despite the biannual vitamin A administration.

Table 4: **Nutritional status of children at the Jimma welfare home, 1995**

Nutritional status	Xeroph. group	Non-Xeroph. group	X^{2*}	p-value
Normal	25	52	1.316	0.251
Wasted	7	14	0.006	0.094
Stunted	6	2	3.895	0.048
Wasted and stunted	1	-	0.08	0.777

* Continuity correction

Table 5: **Disappearance time of XN, XIB and X2, in children at the Jimma welfare home, 1995**

Disappearance time	Number of children	%
Under one week	3(a)	7.69
Between 1 and 2 weeks	10	25.64
Between 2 and 3 weeks	12	30.77
Between 3 and 4 weeks	8(b)	20.51
Between 4 and 6 weeks	2	5.13
Between 6 and 8 weeks	2	5.13
Persistent	2(c)	5.13
Total	39	100.0

(a) all with XN

(b) 2 children with XIB had recurrence

(c) These children had marked shrinkage of Bitot's spots that failed to disappear in toto.

Discussion

This study may remind us of Bloch's observation of children in Danish orphanages where the occurrence of malnutrition and xerophthalmia was associated with consumption of diets deficient in milk and milk products (19). The higher prevalence of clinical VAD among older children may be due to two factors. Firstly, the comparatively higher number of children over seven years of age (72.9% of the study population) might have affected the result. Secondly, the feeding practice at the home is responsible. There are basically three routine feeding profiles noted:

a. children two years and under are fed on different milk formulae (about four to six times a day) that contain considerable amounts of vitamin A; in addition to gruel made from cereals, and, occasionally, eggs.
b. children three to five years of age are fed on mainly porridge made from cereals, milk (about three times a day) and "injera" (special bread made from a tiny seed - "Eragrostis teff") with sauce made from legumes, a variable amount of pepper and oil and, occasionally, eggs.
c. older children (> 5 years of age) are fed almost exclusively on "injera" and sauce.

Foods rich in Vitamin A, like dark-green leafy vegetables (dglu) and fruits, have not reached the home for over two years. Retinol-rich foods like eggs and milk are not available especially for older children.

Kale (Brassica carinata) or "Habesha Gomen" is amply available in and around Jimma and it is not difficult to grow. Papaya, mangoes and carrots are grown in the zone. These vitamin A-rich foods, especially "Habesha Gomen", could be grown at the home to provide the children with some of the nutritive requirement. The information that dark-green leafy vegetables (dglv) may have little to contribute in the prevention of VAD has to be considered seriously.

It implies the need for total revision of the issue of dietary prophylaxis of VAD using dglv. This has again a grim implication for poorer communities that can't afford retinol-rich foods. They may have to depend on the health sector for their periodic supplies of vitamin A megadoses.

The male gender has been associated with a higher risk of developing VAD (4,20-22). No such association was seen in the present study probably because of the small size of the study population. It is known that children with xerophthalmia are prone to diarrhoea and respiratory tract infections (11-14). The lack of association of clinical VAD with morbidity, in the present study, may be attributed to the presence of sub-clinical VAD among some of the non-xerophthalmic children hence masking the possible difference in morbidity. The small population size and morbidity underreporting might also have contributed. Hence, it will be difficult to make conclusions, from this study, regarding morbidity.

Failure of Bitot's spots to disappear completely or their reappearance after some time despite vitamin A supplementation has been documented (23-24). Two children in the xerophthalmic group and one in the non-xerophthalmic group developed Bitot's spots in the second year of initial supplementation. Two of these children had wasting and one was stunted. Other possible causes of the lesions like trauma, trachoma, pemphigus, kerato-conjunctivitis sicca, collagen diseases, etc..were not apparent in these children. Hence other than VAD, malnutrition along with other factors may be responsible for the recurrence of the lesions.

It is recommended that the welfare home administration can tackle the problem through two measures. These are:

1. Short term measures:
    a. improving diet at the home by supplying the kitchen with cheap and locally available sources of the vitamin.
    b. periodic supplementation of mega doses of vitamin A (at least twice a year). This may be done with the help of Jimma eye unit and the Zonal Health Department.
2. Long term measure:

The home should be self-sufficient by developing its own garden. There is adequate land to grow vegetables and fruits.

Acknowledgemtnt

This study was supported by the Research and Publication Office of the Jimma Institute of Health Sciences to which I am grateful. My special thanks goes to Dr. Tekletsion W/mariam for his support and encouragement. I am also thankful to Ato Temam Hussein, Dr. Amha Mekasha, Dr. Afework Assefa, Dr.Tesfaye Getaneh, and Dr. Damtew W/mariam for their contributions to the completion of this work and to Ato Fassil Tessema for the statistical work. The staff at the welfare home deserve my gratitude for their unrelenting cooperation.

References

1. Prevention of Blindness. World Health Statistics Annual. 1990;16-18.
2. Foster A. Childhood Blindness. Eye 1988;2(suppl.):527-36.
3. Thylefors B. The W.H.O's Programme for the Prevention of Blindness. Int'l ophthalmology 1990;14:211.

4. Oomen HAPC. Clinical Epidemiology of Xerophthalmia in Man. Am J Clin Nutr. 1969;22(8):1098-2005.
5. Pizzarello LD. Age specific xerophthalmia rates among displaced Ethiopians. Arch Dis Child 1986; 61:1100-03.
6. De Sole G, Yigzau B, Bekalu Z. Vitamin A deficiency in Southern Ethiopia. Am J Clin Nutr 1987; 45:780-4.
7. Lintjorn B. Xerophthalmia in the Gardula area of South West Ethiopia. Ethiop Med J 1983;21:169-73.
8. Wolde Gebriel Z, West CE, Gebru H, Tadesse A, Fissiha T, Gabre P, et al. Interrelationship between vitamin A, iodine and iron status in school children in Shoa Region, Central Ethiopia.
British J Nutr 1993; 70(2):593-607.
9. De Pee S, West Ce, Muhilal Karyadi D, Hautvast JGA. Lack of improvement in vitamin A status with increased consumption of dark-green leafy vegetables. Lancet 1995;346:75-81.
10. Solomon, NW., and Bulux, J. Plant sources of provitamin A and human nutriture. Nutr Revs 1993; 51:199-204.
11. Sommer A, Katz J, Tarwotjo I. Increased risk to respiratory disease and diarrhoea in children with pre-existing mild vitamin A deficiency. Am J Clin Nutr 1984;40:1090-5.
12. Bloem MW, Wedel M, Egger RJ. Mild vitamin A deficiency and risk to respiratory tract diseases and diarrhoea in preschool and school children in Northeastern Thailand. Am J. Epidemiol 1990;131:332-39.
13. Milton RC, Reddy V, Naidu An. Mild vitamin A deficiency and childhood morbidity-an Indian experience. Am J Clin Nutr 1987;46:827-29.
14. Kothari G. The effect of vitamin A prophylaxis on morbidity and mortality among children in urban slums in Bombay. J Trop Pediatr. 1991;37:141.
15. Arthur P, Kirkwood B, Ross D, Morris S, Jyapong J, Tomking A. et al. Impact of vitamin A supplementation on childhood morbidity in Northern Ghana. Lancet 1992;339:361-62.
16. Ghana VAST Study Team. Vitamin A supplementation in northern Ghana: effects on clinic attendances, hospital admissions, and child mortality. Lancet 1993;342:7-12.

17. Vijayaraghavan K, Radhaiah G, Pranksam BS, Sarma KV, Reddy V. Effect of massive dose Vitamin A on morbidity and mortality in Indian children. Lancet 1990;336:1342-45.
18. Rahmathullah L, Underwood BA, Thulasiraj RD, Milton RC. Diarrhoea, respiratory infections, and growth are not affected by a weekly low dose vitamin A supplement: a masked, controlled field trial in children in Southern India. Am J Clin Nutr 1991;54:568-77.
19. Sommer A. Field Guide for the detection and control of xerophthalmia, W.H.O. 1978.
20. Solon FS, Popkin BM, Fernandez TL, Latham MC. Vitamin A deficiency in the Philippines. A study of xerophthalmia in Cebu. Am J Clin Nutr 1978;31:360-8.
21. Brink EW, Perera WDA, Broske SP, Cash RA, Smith JL, Sauberlich HE. et al. Vitamin A status of children in Sri Lanka. Am J Clin Nutr 1979;32:84-91.
22. Roels OA, Debeir O, Trout M. Vitamin A deficiency in Ruanda Urundi. Trop. Geogr. Med 1958;10: 77-92.3.
23. Sinha DP, Bang FB. The effect of massive doses of Vitamin A on the signs of vitamin A deficiency in pre-school children. Am J Clin Nutr 1976;29:110-115.
24. Sommer A, Emran N, Tjakrasudjatma S. Clinical characteristics of vitamin A responsive and non responsive Bitot's spots. Am J Ophthalmol 1980;90(2):160-71.