

assisting in identifying street addicts and providing staff to fill the questionnaire. Above all, we are grateful to all the intravenous drug users who volunteered to be interviewed.

Nadeem ur Rehman MBBS¹
Faran Emmanuel MBBS MSc²
Saeed Akhtar PhD³

¹Country Office for Pakistan, UNODC;

²Department of Community Health Sciences, Aga Khan University, Stadium Road, Karachi, Pakistan;

³Epidemiology, Department of Community Medicine and Behavioural Sciences, Faculty of Medicine, Kuwait University, PO Box 24923, Safat 13110, Kuwait

Correspondence to: Saeed Akhtar
Email: saeed.akhtar@hsc.edu.kw

References

- 1 UNODC. *Drug Abuse in Pakistan: Results From the year 2000: National Assessment: UNDCP Global Assessment Program on Drug Abuse & Pakistan Anti-Narcotics Force*. 2000. <http://www.unodc.org/>
- 2 Des Jarlais DC, Friedman SR, Choopanya K, Vanichseni S, Ward T. International epidemiology of HIV and AIDS among injecting drug users. *AIDS* 1992;6:1053-68
- 3 Barnard MA. Needle sharing in context: patterns of sharing among men and women injectors and HIV risks. *Addiction* 1993;88:805-12
- 4 Tyndall MW, Patrick D, Spittal P, Li K, O'Shaughnessy MV, Schechter MT. Risky sexual behaviours among injection drugs users with high HIV prevalence: implications for STD control. *Sex Trans Inf* 2002;78:i170-i5
- 5 Royce RA, Sena A, Cates W, Cohen MS. Sexual transmission of HIV. *N Engl J Med* 1992;336:1072-8

Vitamin A deficiency in children under 6 months

Paper presented at the All India Ophthalmologic Society Annual Conference, Varanasi, January 2004

Introduction

Children at highest risk of going blind because of vitamin A deficiency were considered to be those weaned on to solids deficient in vitamin A and who have had an acute increased vitamin A demand due to measles, diarrhoeas, or respiratory infection. Vitamin A deficiency under 6 months is unusual, because newborns have good store of the vitamin, and breast milk contains sufficient quantity. The World Health Organization (WHO)

recommends that children at risk of xerophthalmia receive 100,000 IU of vitamin A with measles vaccine at 9 months of age and subsequently, 200,000 IU, every 6 months, up to 6 years of age.¹ We performed this retrospective analysis of hospital data after it was noticed that there was an unusual increase in the numbers of cases of blinding keratomalacia, in infants less than 6 months of age.

Methods

Records of children with vitamin A deficiency, seen in the hospital over a 9-month period from May 2002 to April 2003 were analysed. Those with acute blinding vitamin A deficiency were studied to evaluate the correlates of the disease. The age of the child, feeding practices, concurrent infection, and the nature and severity of vitamin A deficiency were noted.

Results

Table 1 shows the details of the seven children with corneal xerosis or keratomalacia. Of these, five were children aged 6 months or less. The mean age of the group was 7.57 months, and the median and mode were 4 months (range 2-24 months). In only one (10 months old) was the typical pattern of vitamin A deficiency following measles infection seen. All were born at term except one. Three had bilateral keratomalacia, and another had keratomalacia in one eye and xerosis in the other. None of the infants were breastfed. They were fed dilute, fresh cows milk.

Discussion

In this small sample of children with blinding keratomalacia, 70% cases were aged less than 6 months. This age group is not considered to be at risk for vitamin A deficiency. The National Programme for Prevention of Blindness recommends vitamin A supplementation starting at 9 months of age. This intervention would have been too late for the majority of babies in our study.

West has reported that maternal vitamin A deficiency is a public health problem in developing countries.² Basu *et al.*³ have suggested a mega dose of 209 µmol of retinol be given to mothers at birth and exclusive breastfeeding as means of combating vitamin A deficiency in infancy. The WHO recommends that women receive 10,000 IU daily or 25,000 IU weekly while pregnant, and 200,000 IU after delivery.¹

All this assumes that the baby is breastfed. None of the children in our group was breastfed, and this presents a special challenge. This finding is similar to that of Rahmathullah *et al.*⁴ who observed that 12 of 15 children under 1 year of age with keratomalacia reported by him were not breastfed at all. Bahl *et al.*⁵ suggest vitamin A

Table 1 Details of the babies

Serial number	Age in months	Dietary practices	Manifestation of Vitamin A deficiency	Remarks
1	6	Top fed, dilute cows milk	Right corneal xerosis, left keratomalacia	
2	2	Top fed, dilute cows milk	Bilateral keratomalacia	
3	4	Top fed, dilute cows milk	Right keratomalacia	
4	24	On solid foods	Right keratomalacia	
5	10	Weaning started	Corneal xerosis	Measles
6	4	Top fed, dilute cows milk	Bilateral keratomalacia	
7	3	Top fed, dilute cows milk	Bilateral keratomalacia	Low birth weight baby

supplementation of lactating mothers and of infants at the time of diphtheria-pertussis-tetanus (DPT) and oral polio immunization. This multicentre randomized, double-blind, placebo-controlled trial was conducted in Ghana, India and Peru. Mothers received 60 mg of retinol palmitate at 18–42 days postpartum, and infants received 7.5 mg three times at 6, 10 and 14 weeks. Mother who received the supplement had higher vitamin A in breast milk at 2 months, and less infants were deficient than controls at 6 months. The benefits of this programme were greatest in the Indian cohort studied. Supplementation had no effect at 9 months and the authors suggest additional strategies to improve vitamin A status of 6- to 9-month-old infants. It would appear that a strategy of vitamin supplementation of non-breastfed children must follow the pattern of this study and provide supplements at 6, 10 and 12 weeks.

Ours was a hospital-based study and, from this, it is not possible to estimate the problem in the community. The unusual age at presentation, however, indicates a change in the pattern of the disease, in the community. We need to address the needs of this vulnerable group, if we are to meet the Vision 20/20 by 2020.

Sara Varughese FRCS

Dr Shroff's Charity Eye Hospital, Kedarnath Road,
Daryaganj, Delhi 110002, India
Correspondence to: Dr Sara Varughese
Email: puliyel@vsnl.com

References

- 1 WHO/UNICEF/VAGG/HKI. *Vitamin A Supplements: A Guide to their Use in the Treatment and Prevention of Vitamin A Deficiency*. 3rd edn. Geneva: World Health Organization, 2001
- 2 West Jr KP. Vitamin A deficiency as a preventable cause of maternal mortality in undernourished societies: plausibility and next steps. *Int J Gynaecol Obstet* 2004;**85**:S24-S7
- 3 Basu S, Sengupta B, Paladhi PK. Single megadose vitamin A supplementation of Indian mothers and morbidity in breastfed young infants. *Ostgrad Med J* 2003;**79**:397-402
- 4 Rahmathullah L, Raj MS, Chandravathi TS. Aetiology of severe vitamin A deficiency in children. *Natl Med J India* 1997;**10**:62-5
- 5 Bahl R, Bhandari N, Wahed MA, Kumar GT, Bhan MK. WHO/CHD Immunization-linked Vitamin Group. Vitamin A supplementation of women postpartum and of their infants at immunization alters breast milk retinol and infant vitamin A status. *J Nutr* 2002;**132**:3243-8

Qualitative comparison of qualitative buffy coat and light microscopy in malaria diagnosis

The diagnosis of malaria usually presents a serious challenge to laboratories in most countries, especially in underdeveloped and developing countries where malaria infection is increasing rapidly.

The accepted laboratory practice for the diagnosis of malaria is the preparation and microscopic examination of blood film stained with Giemsa, Wright, or Fields stain.¹ Various rapid, simple, sensitive, and cost-effective diagnostic techniques have been developed to overcome the difficulties encountered in using light microscopy, to

reduce morbidity and mortality especially in children and pregnant patients in most endemic areas.

The qualitative buffy coat (QBC) technique combines an Acridine Orange (AO)-coated capillary tube and an internal float to separate the layer of white blood corpuscles (WBC) and platelets, using centrifugation. Other methods have been claimed to be more sensitive and specific in detection of malaria parasites, than light microscopy.

A total of 270 patients of various ages presenting with clinical symptoms of malaria were examined using QBC and light microscopy. Blood samples were collected aseptically through finger pricking onto clean glass slides for preparation of thin and thick blood films (hereafter, referred to as thick and thin film microscopy (TFM)).

Blood from the antecubital blood veins was collected into a container containing ethylenediaminetetraacetate (EDTA) through vacutainer for easy collection of large volume of blood and to reduce the risk of contamination. Films were examined microscopically after staining with Giemsa and Field's stains, for the presence of malaria parasites. The TFM was considered the 'Gold Standard' for positive results.

For the QBC technique, properly mixed blood was drawn up in the capillary tube provided to a predetermined level indicated by a blue line on the tube. The tube containing a pre-coated AO stain and potassium oxalate was filled with venous blood, and an anticoagulant, which contains a float, was inserted. The tube was centrifuged at 12,000 rpm for 5 min. Components of the buffer coat were separated based on their density due to which they form conspicuous bands.

After centrifugation, the QBC capillary tube was placed on a paraview microscope and examined using a standard light fluorescent microscope equipped with paralens UV microscope adaptor. Fluorescing parasites were observed and identified by the presence of green fluorescent dots at the red blood corpuscles (RBC)/WBC interphase. The results obtained with the TFM were then compared with those of QBC technique, since the TFM method has long been accepted as the Gold Standard for the diagnosis of malaria²⁻⁴

Table 1 shows the percentage distribution of blood samples that were positive and negative using QBC and TFM: 181 (67.0%) were positive for malaria parasite with QBC, whereas 174 (64.4%) were detected to contain malaria parasite using TFM; 89 (33.0%) and 96 (35.6%) gave negative results for QBC and TFM, respectively.

Table 2 shows a high sensitivity of 92.53% and specificity of 79.0%. The positive and negative predictive values were 88.7% and 85.61%, respectively. These figures are inadequate to quantify the suitability of QBC technique. The sensitivity and specificity value of 92.5% and 79.0% can be attributed to the number of samples studied.

Out of the 174 samples that were positive for malaria parasite, 151 (83.4%) and 148 (85.1%) were identified as *Plasmodium falciparum* species through QBC and TFM,

Table 1 Percentage distribution of number positive and negative using QBC and TFM

Method	Number positive (%)	Number negative (%)	Total
QBC	181 (67.0)	89 (33.0)	270
TFM	174 (64.4)	96 (35.6)	270