Diagnosis of night blindness and serum vitamin A level: a population-based study

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In a cross-sectional survey of 5420 children in northern Bangladesh, 124 were reported to have night blindness by their parents. Of these, 105 cases along with controls matched for age, sex, and neighbourhood had their scotopic vision examined under standard conditions using a luxometer, underwent an ophthalmological examination, and had their serum vitamin A level determined. The mean serum vitamin A level was lowest among children identified as night blind by both their parents and the investigators (16.3 µg/dl; 95% confidence interval (CI), 13.9–18.7) and highest among those identified as not night blind by both their parents and the investigators (23.6 µg/dl; 95% CI, 21.3–25.9). The results show that parents' report of their children's night blindness had low sensitivity compared with diagnosis using standard observations of scotopic vision with a luxometer.

Introduction

Vitamin A deficiency is the main cause of irreversible childhood blindness (1–4). Vitamin A is a key precursor in the biosynthesis of rhodopsin (“visual purple”) required by the rods, the retinal photoreceptors responsible for vision under low levels of illumination. Night blindness, also known as hemeralopia and nyctalopia, results from impaired dark adaptation by the light-sensitive retina. Night blindness was previously considered only to be a secondary sign of xerophthalmia and an unreliable criterion for assessing the burden of vitamin A deficiency (5–7).

In recent years, biochemical and histological methodologies have been developed for assessing vitamin A nutritional status in humans at subclinical levels. Application of these specialized methodologies in population-based surveys has, however, been limited because they require expensive instrumentation and there is a lack of trained personnel. Therefore, most field assessments of childhood night blindness have relied on parents' reports of the condition (8–13). However, the validity of this method for assessing the true level of night blindness has recently been the subject of discussion (14, 15).

The objective of the present study was to compare the diagnosis of night blindness based on parents' reports with those based on observation of scotopic vision under standard conditions using a luxometer and on measurements of serum vitamin A concentration in venous blood.

Materials and methods

The study was carried out in Ranjpur district, northern Bangladesh, in 1992. Following a multi-stage random sampling procedure, we selected 2101 households corresponding to 10 757 persons, of whom 5420 were children aged 0–15 years. Parents were interviewed about the occurrence of night blindness among their children. The interviews were conducted in the local dialect. In addition, diagnosis of night blindness was strengthened by posing in-depth questions. Details of the interviewing procedure have been reported previously (13). A total of 124 children aged 2–15 years were registered as having night blindness following interviews with their parents. There were no cases registered among under-2-year-olds. Whenever a case of night blindness was detected, a control was selected from the first neighbouring household that had no child with symptoms of night blindness and who matched the case for sex and age group (2–3, 4–6, 7–8 and 9–15 years). The cases and controls were invited for further examination 1–5 weeks after the interview, and among the investigations carried out were the following: anthropometric measurements, observation of scotopic vision, ophthalmological examination,

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and collection of venous blood for assessment of serum vitamin A level.

A total of 124 cases of night blindness and 124 controls were selected from 105 and 117 households, respectively. Only data from the first case of night blindness in each of the households (105 cases) along with the selected controls (105) are included in the present analyses. Of these, 105 cases and 104 controls had their scotopic vision examined, 103 cases and 104 controls received an ophthalmological examination, and 87 cases and 97 controls had their serum vitamin A level measured (Table 1). The verbal consent of the parents was obtained before the examinations were conducted, and they were informed that they could withdraw their children at any time.

The diagnosis of night blindness was reassessed using the procedure described below. The eyes of the cases and controls were first covered for at least 10 minutes with a piece of dark cloth to induce dark adaptation before each of them was taken into a twilight room equipped with a voltage-regulated electric lamp, a luxometer, and a set of objects of various sizes (diameter from 8–10 mm to 25–30 mm) and colours. Colour contrast was established using different coloured sheets that were laid down on the floor on which the objects were placed. Each child was then told to pick up the smallest objects they could see under twilight conditions. The procedure was repeated at gradually increasing levels of illumination and the degree of night blindness was determined by reading from the luxometer the amount of light (illuminance) needed for the child to pick up all the objects without difficulty. The severity of night blindness was graded as follows: normal, 0–4 lux (level of illuminance necessary for the investigators to identify the objects); and night blind — moderate (5–9 lux); distinct (10–19 lux); severe (≥ 20 lux).

The severity of night blindness and the ophthalmological findings were recorded using a single-blind study design. The children were registered by the ophthalmologist using their household and area number without disclosing their case-control status.

Later these findings were matched with the information collected at the interview.

A sample of 1.5–2.0 ml of free-flow venous blood was drawn from each of the 87 cases and 97 controls using a 3-ml disposable syringe and transferred to a microtube. The tubes were then wrapped with black tape and placed in an ice-bag. Care was taken to avoid shaking the tubes before they were centrifuged. The blood samples were stored in an insulated cool-box for 30 minutes and then centrifuged at room temperature for 15 minutes at 2000 rpm. The sera were separated into another black screw-cap vial, labelled, and frozen at -20°C within 2–8 hours of being collected. Every 10 days, the samples were transported on dry ice to the International Centre for Diarrhoeal Disease and Research, Bangladesh (ICDDR, B), where they were analysed using high-performance liquid chromatography (HPLC) (16).

Statistical comparisons of the different groups were carried out using the χ^2 test, Fisher's exact test and Student's *t*-test. All *P*-values are two-tailed. The Statistical Programme for the Social Sciences (SPSS 4.0) was used for the statistical analysis (17).

Results

Table 2 compares the parents' reports of night blindness with the investigators' diagnosis based on scotopic vision observations using a luxometer. Of the 105 children reported to have night blindness by their parents, only 71 (67.6%) were confirmed as cases by the authors. Of 104 controls, 34 (32.7%) were diagnosed as cases of night blindness by the authors. The observed agreement between the parents' reports and the authors' diagnosis was 67.5% and the estimated κ -statistic was 0.35 ($P < 0.05$).

Serum vitamin A level was lowest among those children identified as night blind cases by both their parents and the investigators (Table 3). Among children for whom there was disagreement about the diagnosis, serum vitamin A was lower among cases

Table 1: Number of cases of night blindness identified by their parents and their matched controls among 5420 children aged 0–15 years, together with their participation in various further investigations, Ranjpure, Bangladesh, 1992

	No. of cases	No. of controls	Total
Total number of cases and controls included in the analysis	105 (100.0) ^a	105 (100.0)	210 (100.0)
Anthropometric measurements	105 (100.0)	105 (100.0)	210 (100.0)
Scotopic vision observation	105 (100.0)	104 (99.1)	209 (99.5)
Ophthalmological examination	103 (98.1)	104 (99.1)	207 (98.6)
Serum vitamin A level assessment	87 (82.9)	97 (92.4)	184 (87.6)

^a Figures in parentheses are percentages.

Table 2: Comparison of the diagnosis of night blindness (XN) made by parents with that based on observation of scotopic vision, Ranjpure, Bangladesh, 1992^a

	Total number of children	No. included in analysis	Scotopic vision diagnosis:	
			XN+ (n)	XN- (n)
<i>Parents' diagnosis:</i>				
XN+	124	105	71 (67.6) ^b	34 (32.4)
XN-	124	104	34 (32.7)	70 (67.3)

^a Agreement between parents' and scotopic vision diagnoses: $\kappa = 0.35$ ($P < 0.05$).

^b Figures in parentheses are percentages.

of night blindness diagnosed by the investigators compared with cases identified by the parents only. The highest serum vitamin A levels were among children classified as not night blind by their parents as well as the investigators.

Of the 104 control children, 22 (21.1%) had moderate, four (3.8%) distinct, and eight (7.7%) severe night blindness according to the luxometer readings (Table 4). For the cases, serum vitamin A level was related to the severity of night blindness ($P = 0.02$), while for the controls the association was weaker ($P = 0.14$).

The odds ratio for the occurrence of night blindness according to the parents' diagnosis was inversely related to the serum vitamin A level. The relationship was stronger when the analysis was restricted to cases and controls confirmed by observation of scotopic vision (Table 5). In these groups, those with serum vitamin A < 10 $\mu\text{g/dl}$ had an odds ratio of 28.9 compared with the reference group with serum vitamin A levels ≥ 30 $\mu\text{g/dl}$. A total of 53 cases (60.9%) and 42 controls (43.3%) according to parents' diagnoses had serum vitamin A levels < 20 $\mu\text{g/dl}$ ($P < 0.05$). Among those whose diagnosis was confirmed by observation of scotopic vision, 71.9% of the cases and 40.9% of the controls had serum vitamin A levels < 20 $\mu\text{g/dl}$ ($P < 0.01$).

Of the 103 children with night blindness according to their parents' diagnosis, 38.8% were found to be suffering from ocular abnormalities upon ophthalmological examination, compared with 26.0%

of the 104 controls ($P = 0.07$) (Table 6). The mean serum vitamin A level was significantly lower in the total series of cases identified by parents compared with that for the controls ($P < 0.01$). Serum vitamin A was particularly low among five cases diagnosed to have Bitot's spots. Cases with retinal changes and keratopathy had also low serum vitamin A levels.

Discussion

A history of night blindness as reported by parents has been used extensively in field studies as a method for assessing the prevalence of the condition. Our findings show that such an approach is less sensitive than standard diagnosis using a luxometer. In one study in Indonesia, parent's diagnosis of night blindness was reported to be sensitive and specific and to have a high positive predictive value in relation to low serum vitamin A levels; it was claimed that the parents' diagnosis was confirmed in 97% of cases by an assessment of scotopic vision (9). However, the validity of parent's reports of night blindness has recently been questioned (14, 15). Cultural diversity, socioeconomic status, and educational and awareness levels of parents are likely to affect the validity of the results. In our study, only 67.6% of the cases reported to be night blind by their parents had this diagnosis confirmed when they were reassessed by the investigators. On the other hand, among the controls (i.e., those not reported to have night blindness by their parents), 32.7% were diagnosed to have night blindness by the authors. As many as 11.5% of the controls had distinct or severe night blindness and required illumination levels ≥ 10 lux before they were able to identify objects.

The lack of agreement between the parents' diagnosis of night blindness and the scotopic visual findings in the studies in Indonesia and Bangladesh could have been due to methodological differences. In Indonesia the observers were not blinded to the children's case or control status and details of the assessment technique used were not described. In a previous study that we carried out, where scotopic vision was observed in twilight/dusk without any

Table 3: Comparison of serum vitamin A level with the diagnosis of night blindness (XN) made by parents and the investigators, Ranjpure, Bangladesh, 1992^a

Diagnosis:			Mean serum vitamin A level ($\mu\text{g/dl}$)
Parents	Investigators	n	
XN+	XN+	57	16.3 (13.9–18.7) ^b
XN-	XN+	31	19.7 (17.5–21.9)
XN+	XN-	30	22.9 (19.7–26.1)
XN-	XN-	66	23.6 (21.3–25.9)

^a Test of homogeneity, $P < 0.001$.

^b Figures in parentheses are the 95% confidence intervals.

Table 4: Distribution of the severity of night blindness among the study children according to parents' diagnosis, the impairment of their dark adaptation, and the estimated mean serum vitamin A level, Ranjpure, Bangladesh, 1992

	Illuminance required ^a (lux)	Parents' diagnosis		Mean serum vitamin A level (µg/l) ^b :	
		Cases (n)	Controls (n)	Cases	Controls
<i>Impairment of dark adaptation:</i>					
Normal	0–4	35	70	22.9 (19.7–26.1) ^c	23.6 (21.3–25.9)
Moderate	5–9	23	22	17.3 (13.9–20.7)	21.1 (18.4–23.8)
Distinct	10–19	26	4	15.7 (10.0–21.4)	18.2 (6.4–30.0)
Severe	≥20	21	8	15.7 (10.9–20.5)	16.6 (12.3–20.9)
Total		105	104	18.6 (16.6–20.6)	22.4 (20.6–24.2)

^a Illumination required for the children to successfully pick up all the test objects.

^b Serum vitamin A level was not measured for 18 cases and 8 controls.

^c Figures in parentheses are the 95% confidence intervals.

standardization and without blinding the observers to the children's status, our findings were similar to those reported for Indonesia (13). The results we have reported in this article show, however, that parents' reports produce a considerable number of false positives. More frequently, however, cases of moderate night blindness diagnosed by the authors were not reported by parents.

Our findings indicate that use of a luxometer is essential to secure an objective assessment of night blindness. With this method, however, care was necessary since room illumination level was difficult to control completely, because the doors, windows, and walls did not fully exclude sunlight. Nevertheless, use of the luxometer permitted an objective assessment of the amount of light in the room, and the method enabled us to classify night blindness according to its severity.

Dark adaptation of the children's eyes by covering them with a piece of black cloth for at least 10 minutes before observations was sometimes diffi-

cult. Identical levels of dark adaptation could not always be realized, since some children were frightened; it is therefore possible that some children did not pick up the objects until the light was bright enough for them to feel secure. To minimize this shortcoming, we recorded the amount of light necessary after making repeated observations, including those made in reverse. To begin with, this was done by encouraging the children either by providing them with sufficient light or with bigger objects to pick up, and then reducing the illumination to the level at which they had difficulty in picking up the smallest objects. Nevertheless, false-positive diagnoses of night blindness could still have been made. However, impairment of scotopic vision, as assessed by our method, was clearly related to the level of serum vitamin A. Children who required illumination levels ≥5 lux had significantly lower serum vitamin A levels than those who required < 5 lux (Table 4). Furthermore, a clear and statistically significant trend was observed between decreasing mean serum

Table 5: Distribution of cases, controls, and odds ratios according to serum vitamin A level, for children diagnosed to have night blindness by their parents and those confirmed by scotopic vision observation, Ranjpure, Bangladesh, 1992

Serum vitamin A level (µg/dl)	Parents' diagnosis:			Confirmed:		
	No. of cases	No. of controls	OR ^a	No. of cases	No. of controls	OR
≥30	10 (11.5) ^b	20 (20.6)	1.0	5 (8.8)	18 (27.3)	1.0
20–29	24 (27.6)	35 (36.1)	1.4; 0.5–3.8 ^c	11 (19.3)	21 (31.8)	1.9; 0.5–7.7
10–19	36 (41.4)	38 (39.2)	1.9; 0.7–5.0	25 (43.8)	25 (37.9)	3.6; 1.0–13.1
<10	17 (19.5)	4 (4.1)	8.5; 1.9–40.7	16 (28.1)	2 (3.0)	28.9; 4.0–152.0

^a OR = odds ratio.

^b Figures in parentheses are percentages.

^c Figures in italics are the 95% confidence intervals.

Table 6: Serum vitamin A levels according to the findings of ophthalmological and ophthalmoscopic examinations for cases of night blindness and controls (parents' diagnosis), Ranj-pure, Bangladesh, 1992

	No. of cases	Mean serum vitamin A level ($\pm$ SD) (μ g/dl)	No. of controls	Mean serum vitamin A level ($\pm$ SD) (μ g/dl)
<i>Ophthalmological diagnosis:</i>				
Injection	5	22.1 $\pm$ 6.7	2	18.7 $\pm$ 0.2
Dryness/wrinkling of conjunctiva	3 (1) ^a	18.8 $\pm$ 11.8	3 (1)	19.8 $\pm$ 15.3
Diffuse pigmentation of conjunctiva	15 (1)	17.1 $\pm$ 9.1	16 (2)	24.4 $\pm$ 8.0
Bitot's spots	5	8.8 $\pm$ 9.3	0	
Foam on cornea/conjunctiva	1	23.8	0	
Keratopathy	6 (1)	14.9 $\pm$ 7.1	0	
Others	0		6	24.5 $\pm$ 10.7
<i>Ophthalmoscopic diagnosis</i>				
Retinal changes/white retinal specks	3	11.3 $\pm$ 6.8	0	
Myopic fundus	2	39.8 $\pm$ 8.7	0	
Total abnormalities	40	16.5 $\pm$ 8.9	27	23.6 $\pm$ 8.7
No observed abnormalities	63 (13)	19.8 $\pm$ 9.7	77 (4)	22.0 $\pm$ 9.1
Total	103 (16)	18.6 $\pm$ 9.4	104 (7)	22.4 $\pm$ 9.1

^a Figures in parentheses are the number of cases and controls whose serum vitamin A level was not determined.

vitamin A level, and increasing severity of night blindness among the cases and controls (parents' diagnosis) ($P < 0.001$).

We carried out our assessment of night blindness 1–5 weeks after the initial survey. This procedure was adopted since it would have been difficult to have had the services of an ophthalmologist and of biochemistry technicians, as well as the necessary instruments and a power generator, over a long period of time. Moreover, in rural areas of Bangladesh collecting blood samples from children could have frightened a considerable number of inhabitants, resulting in possible underreporting of cases of night blindness. Some of the children reported by their parents to have night blindness might have recovered over the assessment period because of changes in their diet or the use of vitamin A capsules; however, it is unlikely that approximately one-third of the children would have done so over this period. Similarly, it is unlikely that 34 of the 104 controls not reported to have night blindness by their parents would have developed the condition in this 1–5-week period.

Parasitic infections can influence vitamin A status (18). The cases and controls were matched for age, sex and neighbourhood, thereby ensuring that the socioeconomic conditions and environmental factors that could influence the occurrence of disease were comparable for cases and controls. Moreover, the proportion of children reported to have suffered from an illness in the 2 weeks prior to their examination was similar for cases (49%) and controls (48%), and it is therefore unlikely that the results were

markedly influenced by differences in the occurrence of infectious diseases among cases and controls.

The prevalence of night blindness among the controls was probably higher than that among a random sample of children in the study area, since the controls were matched for residence, and lived in a neighbourhood with a higher than average prevalence of night blindness. Nevertheless, the true prevalence of night blindness in this community is probably considerably greater than the estimate based on parents' diagnosis only. Thus, parents' reports of a history of night blindness can only provide a crude estimate of the prevalence in a community.

Determination of serum vitamin A level is often used to assess the severity of hypovitaminosis A in a community. However, such measurements are expensive, time-consuming, and require trained personnel as well as sophisticated instrumentation. Among the children selected as controls and whom we confirmed not to be night blind, 41% had serum vitamin A levels $< 20 \mu$ g/dl, which is considered to be low (10). A large proportion of the children in the study area therefore have chronic low serum vitamin A levels and hence are at high risk of developing night blindness and other signs of xerophthalmia. This finding is also in agreement with studies of serum vitamin A levels in other areas with a high prevalence of night blindness. For example, a study in India (19) showed that the mean serum vitamin A level among children who were not night blind was 17.5μ g/dl, while in Indonesia 45% of children reported not to be night blind and who did not have

Bitot's spots had serum vitamin A levels $<20 \mu\text{g/dl}$ (9). These findings support the need for a simple and sensitive method for early detection of night-blind children.

Five of the 57 confirmed cases of night blindness had normal serum vitamin A levels ($\geq 30 \mu\text{g/dl}$). All these five children were aged 9–15 years. It has previously been suggested that night blindness may be the earliest clinical sign of vitamin A deficiency, and that it can occur at serum vitamin A levels $>20 \mu\text{g/dl}$ (8, 20, 21). For example, a study in Brazil reported that 21% of children with initial serum levels $>30 \mu\text{g/dl}$ had positive dose-response tests, indicating inadequate vitamin A status (22). However, the odds ratio for the prevalence of night blindness was inversely related to serum vitamin A level, particularly at levels $<10 \mu\text{g/dl}$ (Table 5). These findings are in agreement with those reported by the International Vitamin A Consultancy Group (IVACG) (23), showing that, in most cases, serum vitamin A levels $<10 \mu\text{g/dl}$ perturb scotopic vision. In our study, the association was stronger for the cases reported by parents and which we subsequently confirmed, indicating that our diagnosis was more strongly related to serum vitamin A level.

Ophthalmological examination revealed significantly more abnormalities among the cases reported by the parents than among the controls. Of 40 cases diagnosed with abnormalities, only 17 were associated with signs of vitamin A deficiency (Bitot's spots, dryness of conjunctiva, keratopathy, and retinal changes). This suggests that some of the children reported by their parents to have night blindness could have been suffering from refractive or other anomalies. Although the mean serum vitamin A level for the cases with abnormalities was $16.5 \mu\text{g/dl}$, not all of them showed signs of vitamin A deficiency. Our findings showed that low levels of serum vitamin A are often not related to clinical signs, which is in agreement with previous studies (24, 25).

Carrying out impression cytology in a rural setting is not straightforward. The children, particularly the younger ones, were sometimes frightened and this reduced their cooperation. No child was, however, forced to participate in this part of the study. Also, fixing and straining the collected conjunctival cells on a glass-slide in a highly humid and dusty milieu was complicated, most specimens collected contained few cells, and the presence of goblet cells could not be reliably assessed. It has been reported that conjunctival impression cytology is not particularly successful with children who are crying, is difficult to perform on under-3-year-olds, and has a low sensitivity in dry climates (26). This diagnostic approach may therefore not be suitable for use under field conditions.

A reliable method for accurate assessment of the prevalence of night blindness in a population is not yet available. However, the method we employed in this study is simple, does not require highly trained personnel, is not expensive, and can be used without access to a supply of electricity. The method should, however, be tested in different settings to assess its validity and repeatability. Our results suggest that the extent of the problem of night blindness assessed solely using parents' histories represents a crude estimate and should therefore be interpreted with caution.

In India, attempts have been made to develop a new field test to identify night blindness (19). The methodology is based on registering the duration of dark adaptation after subjects have been exposed to three consecutive flashes in a dark room, with the test being repeated on three successive days. This procedure, however, has obvious limitations: it requires access to a supply of electricity, use of a camera flash-gun is likely to frighten a considerable proportion of the children (resulting in an inaccurate determination of the dark adaptation time), carrying out observations on separate days may be difficult, and it may not be suitable for under-7-year-olds.

Although the estimated prevalence of night blindness based on parents' diagnosis is a crude indicator of the severity of the problem in a population, it can still have value provided the sensitivity and specificity of the parents' diagnosis do not change much over time. Under these circumstances, this approach can still be used to identify populations in need of intervention programmes and to provide a crude assessment of the prevalence of night blindness over time in a given population. Future studies of the prevalence of night blindness should employ a standard method of observing random samples of children. The present study showed that the use of a luxometer in addition to parents' histories of night blindness increased the sensitivity of the diagnosis, and that, if possible, serum vitamin A levels should also be determined.

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Résumé

Diagnostic de la cécité nocturne et vitamine A sérique: étude en population

Il n'existe pas encore de méthodes simples et valables de diagnostic de la cécité nocturne. Cette situation peut influencer les résultats des études de prévalence et l'évaluation des variations de celle-ci au cours du temps et selon les régions. Lors d'une enquête transversale portant sur 5420 enfants à Ranjpur, Bangladesh, 124 d'entre eux étaient, selon leurs parents, atteints de cécité nocturne; 105 de ces cas ont été inclus dans l'étude et ont été appariés avec des témoins selon l'âge, le sexe et le lieu de résidence. La vision scotopique a été évaluée dans des conditions normalisées au moyen d'un luxmètre. Tous les cas et témoins ont été en outre soumis à un examen ophtalmologique et à un dosage de la vitamine A sérique. Le diagnostic n'a été confirmé par les investigateurs que pour 67,6% des cas signalés par leurs parents; sur 104 témoins examinés, les investigateurs ont diagnostiqué 32,7% de cas de cécité nocturne. Le taux moyen de vitamine A sérique était le plus faible dans un groupe de 57 enfants chez qui le diagnostic de cécité nocturne avait été fait à la fois par les parents et par les investigateurs (16,3 µg/dl; intervalle de confiance à 95% (IC): 13,9–18,7), et le plus élevé chez 66 enfants indemnes de cécité nocturne d'après leurs parents comme d'après les investigateurs (23,6 µg/dl; IC: 21,3–25,9). Le odds ratio pour la cécité nocturne augmentait significativement en raison inverse du taux sérique de vitamine A et était particulièrement élevé chez les enfants ayant un taux de vitamine A <10 µg/dl. La mention d'une cécité nocturne par les parents était peu sensible et peu spécifique par rapport au diagnostic reposant sur un examen normalisé de la vision scotopique au moyen d'un luxmètre. Nous suggérons en conséquence d'inclure cet examen dans les enquêtes de terrain portant sur la prévalence de la cécité nocturne.

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