Vitamin A deficiency and corneal ulceration in south-east Nepal: implications for preventing blindness in children

A. Hennig, A. Foster, S.P. Shrestha, & R.P. Pokhrel

A retrospective review of the outpatient records of 4601 children aged 0–10 years who had been seen between January 1986 and December 1988 at Lahan Eye Hospital, south-east Nepal, revealed that 15.4% had evidence of active or past xerophthalmia. Of 293 children with corneal xerosis or corneal ulcer, 49% had been examined in the 4-month period May–August. The peak age for active noncorneal xerophthalmia was 5 years and for active corneal xerophthalmia, 3 years.

Previous population-based studies in Nepal have documented the presence of noncorneal xerophthalmia (Bitot’s spots) in children. The present study confirms that vitamin A deficiency is a major cause of blindness and loss of vision among children in the eastern plains of Nepal.

Introduction

Globally, xerophthalmia is the principal cause of childhood blindness (1). Two population-based studies in Nepal have shown that Bitot’s spots are common, occurring in 1.65% of 6118 children aged 0–14 years (2) and 0.64% of 7580 children aged 0–6 years (3), which suggests that vitamin A deficiency is a public health problem in the country. However, among the 13698 children examined in both these studies combined, only one child with active corneal xerophthalmia was observed.

We therefore conducted a retrospective review of the hospital records of all children who attended a busy eye hospital in the south-east plains area of Nepal, to document the occurrence and characteristics of cases of active noncorneal and corneal xerophthalmia.

Methods

The outpatient cards of all new patients aged 0–10 years who were examined between January 1986 and December 1988 at Lahan Eye Hospital were reviewed. Active noncorneal xerophthalmia was defined as the presence of Bitot’s spots with conjunctival xerosis (X1A + X1B). Bitot’s spots without conjunctival xerosis were not included in the definition, since it is impossible without treatment to determine whether such spots are evidence of active or of previous vitamin A deficiency. Also, conjunctival xerosis alone (X1A) was not included because of the unreliability of this sign. All children with corneal ulcer or corneal xerosis attributable to vitamin A deficiency, according to clinical appearance and response to vitamin A therapy, and who did not have other obvious causes of corneal ulcer, e.g., trauma or infection with herpes simplex virus, were included and defined as cases of active corneal xerophthalmia (X2 or X3). The following information was recorded for each child: age, sex, month of presentation, association with other systemic diseases, and place of residence.

Results

Of the 4601 children aged 0–10 years seen at the hospital over 3 years, 176 (3.8%) had conjunctival xerosis in the presence of Bitot’s spots; a further 78 had corneal xerosis and 217, corneal ulcer thought to be due to vitamin A deficiency (6.4% combined). A total of 159 children (3.5%) had corneal scar attributable to previous xerophthalmia and an additional 80 children (1.7%) had Bitot’s spots alone, which could have been due to past or present vitamin A deficiency (Table 1).

There was a seasonal variation among cases, with 43% (75 of 176) of noncorneal xerophthalmias and 49% (145 of 295) of corneal xerophthalmias being seen between May and August (Fig. 1 and Fig. 2).
Table 1: Clinical presentation of xerophthalmia by age and sex, among children seen at Lahan Eye Hospital, Nepal, 1986–88

<table>
<thead>
<tr>
<th>Age (years)</th>
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<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Total number of children</td>
<td>1285</td>
<td>909</td>
<td>1523</td>
<td>884</td>
<td>2808</td>
<td>1793</td>
<td>4601</td>
<td></td>
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</tr>
<tr>
<td>No. with active noncorneal xerophthalmia (X1A + X1B)</td>
<td>77</td>
<td>40</td>
<td>39</td>
<td>20</td>
<td>116</td>
<td>60</td>
<td>176</td>
<td></td>
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<tr>
<td>No. with active corneal xerophthalmia (X2)</td>
<td>45</td>
<td>25</td>
<td>5</td>
<td>3</td>
<td>50</td>
<td>28</td>
<td>78</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>(X3)</td>
<td>108</td>
<td>89</td>
<td>12</td>
<td>8</td>
<td>120</td>
<td>97</td>
<td>217</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. with previous xerophthalmia (XS)</td>
<td>62</td>
<td>62</td>
<td>18</td>
<td>17</td>
<td>80</td>
<td>79</td>
<td>159</td>
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</table>

* Another 80 children had Bitot’s spots alone (X1B), which could have been due to past or present vitamin A deficiency

Fig. 1. Illustration of the seasonal variation in the number of children who presented with active noncorneal xerophthalmia (X1A + X1B), Lahan Eye Hospital, Nepal, 1986–88.

The peak age for noncorneal xerophthalmia was 5 years, while the peak incidence for corneal xerophthalmia (corneal xerosis and corneal ulcer) was among 3-year-olds (Fig. 3 and Fig. 4).

Evidence of noncorneal xerophthalmia was observed in 4.1% of males compared with 3.3% of females, and signs of corneal xerophthalmia in 6.1% of males compared with 7.0% of females.

A total of 85% of the children with corneal ulcers had had a history of diarrhoea that lasted at least 3 days in the month prior to being examined at the hospital. Of the children with active corneal xerophthalmia, 56% (164 of 295) came from Sagarmatha Zone, 23% (68) from Janakpur Zone, 5% (16) from other zones, and 47 (16%) from India (Fig. 5).

Discussion

Xerophthalmia in Nepal

Previous population-based studies in Nepal have shown that xerophthalmia is a public health problem. Upadhyay et al. surveyed 6118 children aged 0–14 years and found the overall prevalence of Bitot’s spots to be 1.65% (95% confidence interval, 1.32–1.98%), that of corneal ulcers, 0.02%, and corneal scarring, 0.03% (2). In the Nepal Blindness Survey carried out in 1981, 7580 children under 6 years of age were examined. The prevalence of conjunctival xerosis was 0.22%, that of Bitot’s spots, 0.64%, and of corneal scarring, 0.2% (3). Administratively, Nepal is divided into the following regions; the far west, west, central, and eastern. The
Vitamin A deficiency and corneal ulceration in south-east Nepal

Fig. 2. Illustration of the seasonal variation in the number of children who presented with active corneal xerophthalmia (X2 or X3), Lahan Eye Hospital, Nepal, 1986–88.

![Bar chart showing seasonal variation in the number of children with corneal xerophthalmia](chart1.png)

Fig. 3. Number of patients with noncorneal xerophthalmia, stratified by age.

![Bar chart showing age distribution of patients with noncorneal xerophthalmia](chart2.png)

prevalences of Bitot’s spots in these regions reported by Upadhyay et al. and by the Nepal Blindness Survey are shown in Fig. 5 (2, 3).

However, in these two surveys only one child was seen with an active corneal ulcer caused by vitamin A deficiency. This child came from the hills in the western region of the country and was aged 4.5 years (2). Two previous hospital-based studies carried out in 1978–79 (4) and 1975–80 (5) reported 20 and 73 cases of xerophthalmia, respectively, but identified only one child with corneal ulcer and six other children with corneal xerosis. Thus, although there is good evidence from population and hospital-based studies for the presence of Bitot’s spots in Nepalese children, there are few documented cases of active blinding corneal xerophthalmia.

Over the period January 1986 to December 1988, 295 children with corneal xerophthalmia were seen at Lahan Eye Hospital in south-east Nepal. Most of these children came from the plains area, were malnourished, and had recently suffered a diarrhoeal illness. Of children with corneal ulcers, 87% were aged 1–5 years, and 49% with active corneal xerophthalmia presented in the period May–August. March–May is the period when fresh fruit and vegetables are scarcest, while the monsoons begin in May, with a consequent increase in childhood diarrhoea. The combination of low vitamin A intake and diarrhoea at this time of the year causes the increased incidence of corneal ulcer from vitamin A deficiency.

In Nepal, short-term measures to prevent vitamin A deficiency should therefore target 1–5-year-olds and concentrate on the administration of vitamin A supplements during the premonsoon period (March–June), particularly to children from...
poor, low-caste families in the central and eastern plains (3). The long-term strategy to prevent xerophthalmia should consist of nutrition education aimed at improving the dietary vitamin A intake of preschool-age children. The prevention and early management of diarrhoeal diseases will also help to reduce the incidence of corneal xerophthalmia.

**Childhood blindness in Nepal**

The overall prevalence of blindness found by Upadhyay et al. among children aged 0–14 years in Nepal was 1.14 per 1000; bilateral corneal scars or phthisis bulbi occurred in three of seven blind children, while three others had congenital cataracts (2). In the Nepal Blindness Survey, 11 out of 17279 children aged 0–14 years were blind—a prevalence of 0.64 per 1000: two children were blind because of vitamin A deficiency, two as a sequela of measles, and two from congenital cataracts. Xerophthalmia was held to be responsible for 18% of childhood blindness (3).

The results of these two studies suggest that vitamin A deficiency is responsible for 18–43% of childhood blindness in Nepal. This is, however, probably an underestimate of the true extent of the problem posed by blindness from xerophthalmia in Nepal for the reasons outlined below. First, it is difficult to ascribe definitively corneal blindness in children to vitamin A deficiency rather than other causes of corneal disease, e.g., infection or trauma.

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**Fig. 4. Number of patients with corneal xerophthalmia, stratified by age.**

![Bar chart](image)

**Fig. 5. Map of Nepal showing the prevalence of Bitot’s spots (per 100) found in two population-based surveys.**

<table>
<thead>
<tr>
<th></th>
<th>Far west</th>
<th>West</th>
<th>Central</th>
<th>East</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upadhyay et al. (6118 children: 0-14 years)</td>
<td>2.97</td>
<td>1.81</td>
<td>1.72</td>
<td>0.14</td>
</tr>
<tr>
<td>Nepal Blindness Study (7580 children: 0-5 years)</td>
<td>0.9</td>
<td>0.5</td>
<td>0.4</td>
<td>1.2</td>
</tr>
</tbody>
</table>
Cases of corneal blindness caused by vitamin A deficiency may be attributable to measles or be of unknown cause (3). Second, mortality from vitamin A deficiency and blindness in young children is high. For example, in Bangladesh, Cohen estimated that at least 50% of children blinded by vitamin A deficiency died within a year (6). Estimates of the number of children blinded by xerophthalmia should therefore be viewed with caution since they are based on cross-sectional prevalence data, while the true level can only be obtained from incidence data.

Conclusions
The findings of this retrospective hospital-based study in the plains area of south-east Nepal are complementary to two previous population-based surveys (2, 3), which reported that Bitot’s spots were common in Nepalese children—although only one child with a corneal ulcer was identified. This is to be expected, since corneal ulceration lasts for only a few days or weeks, and cross-sectional surveys are therefore unlikely to encounter many children with the acute disease (especially if the surveys are conducted between September and May, when active xerophthalmia is least frequent in Nepal).

On average, over the last 3 years, two children per week with potentially blinding xerophthalmia have been seen at Lahan Eye Hospital. The results of our study support the data from population-based surveys that xerophthalmia is a public health problem in the plains area of east Nepal, for which urgent action is required to prevent further preventable childhood blindness.

Acknowledgements
We thank the Christoffel Blindenmission, for supporting this study, and the Nepal Prevention of Blindness Programme, Ministry of Health, Nepal.

Résumé
Carence en vitamine A et ulcérations cornéennes dans le sud-est du Népal: conséquences pour la prévention de la cécité chez les enfants

Un examen rétrospectif des dossiers ambulatoires de 4601 enfants âgés de 0 à 10 ans qui avaient été vus au Lahan Eye Hospital, dans le sud-est du Népal, entre janvier 1986 et décembre 1988, a montré que 15,4% avaient des signes de xérophthalmie active ou ancienne. Sur les 293 enfants atteints de xérosis cornéen et d’ulcérations cornéennes, 49% avaient été examinés dans la période de quatre mois allant de mai à août. Le pic d’âge pour la xérophthalmie active non cornéenne était de 5 ans et pour la xérophthalmie cornéenne active, de 3 ans. L’étude confirme que la carence en vitamine A est une cause importante de cécité et de perte de la vision chez les enfants des plaines orientales du Népal.

Nos résultats s’ajoutent à ceux des enquêtes précédentes de population, qui ont rapporté que les taches de Bitot étaient fréquentes chez les enfants népalais, bien qu’un seul enfant atteint d’ulcérations cornéennes ait été identifié. Cela était à prévoir du fait que les ulcérations cornéennes ne durent que quelques jours ou quelques semaines. Des enquêtes transversales ont de ce fait peu de chances de retrouver de nombreux enfants atteints de xérophthalmie aiguë, particulièrement si les enquêtes sont effectuées entre septembre et mai, période à laquelle la xérophthalmie active est la moins fréquente au Népal.

Pendant les trois dernières années, une moyenne de deux enfants par semaine ont été vus au Lahan Eye Hospital pour une xérophthalmie pouvant être une cause de cécité. Nos résultats corroborent ceux des enquêtes de population et ont montré que la xérophthalmie est un problème de santé publique dans la région des plaines du Népal oriental et qu’il est urgent de prendre des mesures permettant de prévenir les cas de cécité évitable chez l’enfant.

References