Conjunctival Appearance in Corneal Xerophthalmia

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I It is widely believed that xerophthalmic corneal destruction occurs in clinically "white and quiet" eyes in which the conjunctiva is distinctly xerotic. Our observations indicate that this is frequently not the case. Xerophthalmic ulceration and necrosis are commonly accompanied by inflammation; conjunctival injection may mask background xerosis; and in severe precipitous deterioration of vitamin A status, clinically recognizable alterations of the cornea sometimes developed before any changes appeared in the conjunctiva.

(Arch Ophthalmol 1962;100:951-952)

SUBJECTS AND METHODS

Between June 1977 and September 1978, a total of 162 consecutive children were seen at the Cendro Eye Hospital, Bandung, Indonesia, with gross nutritional keratopathy. In 53 of the cases, the severest lesion was corneal xerosis (X2); in 50, small eccentric ulcers (X3A) in 31, larger areas of focal necrosis (X3B); and in 25, complete corneal destruction of one or both eyes (X3B). The corneal changes in these cases have already been described. This serum level of bilirubin-binding protein was severely depressed in all subjects, and the serum vitamin A level was inversely related to the degree of corneal involvement. In most instances, this level of vitamin A was distinctly xerotic (Table 2).

The 162 cases of X2 and X3 contained 216 eyes with frank active corneal involvement. Conjunctival injection, often severe, was present in 132 (47%). Injection was more prevalent in eyes with stromal loss (X5) than in those with isolated corneal xerosis (Table 1) (P < .001). Conjunctival xerosis was recognized in 101 (95%) of the 106 eyes of cases with X2 in at least one eye, but in only 152 (70%) of 216 eyes of cases with X3 in at least one eye (P < .001). Among the latter cases, conjunctival xerosis was more prevalent in nonulcerated eyes (53/61; 87%) than in eyes with corneal ulceration of necrosis (59/155; 38%) (P < .01). Two eyes of patients with X3 are omitted from the analysis: one was destroyed by a previous episode of xerophthalmia, and the record of the other failed to note the presence or absence of conjunctival xerosis.

Conjunctival xerosis was just as extensive in nonulcerated eyes of patients with X3 as in eyes of cases of corneal xerosis. It was more extensive in both than it was in cases of isolated vitamin A-responsive conjunctival xerosis (Table 2).

In 21 of the 106 subjects with corneal involvement, conjunctival xerosis was present in one eye but not the other. The nonxerotic eye was far more likely to be injected, and its cornea more severely involved, than its xerotic mate (Table 3).

Clinically apparent involvement of the cornea sometimes preceded that of the conjunctiva, particularly when vitamin A status deteriorated rapidly. One such patient's course is detailed in the following section.

REPORT OF A CASE

A 2-year-old boy with severe generalized malnutrition had bilateral conjunctival xerosis and X2 and an inferior ulcer in the left eye. Because his parents refused to allow hospitalization, he received only a single dose of 200,000 IU of oil-soluble vitamin A. Despite the persistence of severely decreased levels of albumin (< 1.7 g/dL) and transferrin (< 25 mg/dL), both eyes healed within nine days, bilirubin-binding protein and vitamin A levels peaked during this interval at 12 μg/mL and 32

Table 1.—Prevalence of Conjunctival Injection in Eyes With Nutritional Keratopathy

<table>
<thead>
<tr>
<th>Severity of Corneal Involvement</th>
<th>No. of Eyes</th>
<th>No. (%) of Eyes With Conjunctival Injection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xerosis</td>
<td>143</td>
<td>21 (15)</td>
</tr>
<tr>
<td>Ulcer(s)</td>
<td>72</td>
<td>52 (72)</td>
</tr>
<tr>
<td>Focal necrosis</td>
<td>44</td>
<td>34 (77)</td>
</tr>
<tr>
<td>Complete necrosis</td>
<td>37</td>
<td>25 (68)</td>
</tr>
<tr>
<td>Total</td>
<td>296</td>
<td>132 (44)</td>
</tr>
</tbody>
</table>

*Presence or absence of injection was recorded in two eyes.

Table 2.—Extent of Conjunctival Xerosis (X1) in Eyes of Ophthalmic Pattrients

<table>
<thead>
<tr>
<th>Patient Classification</th>
<th>Total No. of Eyes</th>
<th>Extent of X1. No. (%) of Eyes</th>
</tr>
</thead>
<tbody>
<tr>
<td>X1</td>
<td>100</td>
<td>5(5)</td>
</tr>
<tr>
<td>Corneal xerosis (X2)</td>
<td>106</td>
<td>5(5)</td>
</tr>
<tr>
<td>Nonulcerated fellow eye of X3 cases</td>
<td>61</td>
<td>8(13)</td>
</tr>
</tbody>
</table>

*Omits eyes for which record failed to note the presence or absence of X1. Difference in extent of X1 among cases with and without corneal involvement was P < .001.
In a small number of cases, precipitous deterioration of vitamin A status resulted in clinically recognizable alterations of the cornea preceding those of the conjunctiva, thus reversing the usual sequence. This provides at least one mechanism by which nutritional keratopathy can appear in the absence of both conjunctival xerosis and inflammation.

In differentiating nutritional keratopathy from infectious or other forms of corneal destruction, the presence of conjunctival xerosis is far more helpful than its absence. Clinically significant vitamin A deficiency should be suspected whenever X2, X3A, or X3B are encountered in an appropriate dietary, social, or medical setting, regardless of the appearance of the conjunctiva.

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**References**