Herpetic canalicular obstruction

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SUMMARY Viral infection is a common cause of acquired obstruction of the lacrimal canalicular system. A series of 20 patients with canalicular obstruction attributable to infection with herpes simplex is reported, and 1 case is described in detail.

The purpose of this paper is to describe the pattern of disease in the lacrimal drainage system resulting from infection with herpes simplex virus and to discuss the approach to investigation and treatment. A large proportion of canalicular blocks are attributable to viral infections of the external eye, and herpes simplex virus is an important pathogen in this regard. In recent years we have treated 20 patients in whom we are confident the cause of the acquired canalicular obstruction was herpes simplex virus. Here we review these cases as a group and report 1 typical case in detail.

Materials and methods

Twenty patients presented with evidence of herpetic disease and epiphora. Seventeen were female, 3 were male, and all were in the first 3 decades of life when they initially developed epiphora. The earliest onset of epiphora was at age 3 months and the latest at 26 years (Table 1).

In all cases the onset of epiphora was related to an episode of unilateral conjunctivitis associated with lid vesicles, although in some cases years passed before advice was sought. Five patients presented during this initial episode, and a diagnosis of primary herpes simplex blepharokeratoconjunctivitis was made. In others the primary attack had long subsided before the patient realised the epiphora was to be a permanent sequel to the initial infection.

Eleven patients developed recurrent dendritic ulceration, and 4 others had corneal scarring consistent with healed herpetic keratitis, although they denied symptoms. In 3 cases positive isolates of herpes simplex virus were obtained, although no active herpetic process was apparent. The pattern of herpetic disease in the external eye is set out in Table 2.

| Table 1 Distribution of the ages of 20 patients presenting with epiphora attributable to infection with herpes simplex virus |
|---|---|---|---|---|---|
| 0-- | 5-- | 10-- | 15-- | 20-- | 25--29 |
| 3 | 8 | 3 | 5 | 0 | 1 |

| Table 2 Manifestations of herpetic disease in 20 patients with canalicular obstruction attributable to herpes simplex infection |
|---|---|
| Manifestation of herpetic disease | No. of patients |
| Primary blepharokeratoconjunctivitis | 20 |
| Recurrent dendritic ulceration | 11 |
| Corneal scarring without symptoms | 4 |
| Herpes simplex virus isolated | 3 |

The site of the canalicular pathology was remarkably consistent, the midzone being affected in all cases and the proximal end of the block being 2 to 6 mm beyond the puncta, which were invariably normal. The upper and lower canaliculi were affected in each case. Only 9 of the 20 cases had been treated with 5-ido-2'-deoxyuridine (IDU) prior to the development of epiphora.

Biopsy material was obtained from 3 cases at the time of bypass surgery. Light microscopy showed areas of fibrosis with mononuclear cell infiltration, and with electron microscopy particles which could represent herpes simplex virus were seen (Fig. 1). The size of the particles is consistent with their being herpes simplex virus, as is the shape—round with a light core and dark outer zone.

Treatment

Midcanalicular disease is not amenable to plastic reconstructive surgery, and in all cases it eventually proved necessary to perform a bypass procedure
with a Lester Jones tube to cure the epiphora. The longest follow-up period was 7 years.

Case report

A 35-year-old man presented with epiphora which he had had for 10 years. The onset of symptoms coincided with a 2-week episode of unilateral conjunctivitis associated with lid vesicles. He had subsequently suffered several episodes of dendritic ulceration of the cornea.

The lacrimal puncta appeared normal, the sac was not palpable, and syringing revealed a patent upper canaliculus, but the lower canaliculus was obstructed 3 mm beyond the punctum. The conjunctiva was normal, but the cornea was scarred centrally. Intubation macrodacryocystography confirmed the blocked lower canaliculus and a patent upper canaliculus, which was irregular in the mid-zone (Fig. 2).

To identify herpes simplex virus positively as the cause of the disease is more difficult. Virus isolation studies were negative, complement fixing antibodies were demonstrated at a 1:20 titre, but herpes simplex

Fig. 1 Electronmicrograph of excised area of occluded lacrimal canaliculus. The round structures with a dense periphery have a size and morphology consistent with herpes simplex virus
virus antibodies are found in 75% of the community (Andrewes and Carmichael, 1930). This level is indicative of exposure to antigen at some point and is consistent with a primary infection 10 years previously.

Initially an attempt was made to perform a canalicularostomy, but this failed to control the epiphora. Because only the lower canalicus was obstructed and the upper canalicus was apparently patent, a dacryocystorhinostomy was performed. This procedure is often successful when only 1 canalicus is patent (Jones and Corrigan, 1969). However, in this case the epiphora persisted, presumably because the upper canalicus, although patent, is quite irregular and unable to function normally. Only when a Lester Jones tube was inserted did the patient become symptom-free.

**Discussion**

Viral infections are a well-recognised cause of acquired canalicular obstruction (Bouzas, 1965; Sandford-Smith, 1970; Wise, 1976; Welham, in press). They constitute the second most common cause of acquired canalicular obstruction in our practice. A breakdown of the causes of canalicular obstruction treated surgically over a 10-year period to 1976 is set out in Table 3. Other viral diseases are known to cause canalicular obstruction, but in patients treated in the Lacrimal Clinic at Moorfields Eye Hospital, City Road, London, herpes simplex virus was the most common.

The diagnosis of herpetic canalicular obstruction

in the 20 cases reported here was based on clinical findings and laboratory investigations. The major obstacle to confirming the clinical diagnosis with virus isolations is the long period between the occurrence of the active disease process and the presentation with epiphora. Almost always the disease is quiescent by the time the patient realises the watering is a permanent sequel to the inflammation of the external eye.

The importance of the primary infection with herpes simplex virus is emphasised by the fact that all patients dated their symptoms from an episode of vesicular blepharokeratoconjunctivitis and the youth of those afflicted. The remarkable similarity in the clinical features exhibited in this group may be due to our preparedness to recognise these more obvious cases. No cause is apparent in most cases of nontraumatic acquired canalicular obstruction. Perhaps within this group infective causes are important but are less obvious in their manifestations. That both canaliculi are affected in all 20 cases suggests that we are looking at the more severe end of the spectrum, and other patients with herpetic infections of the external eye may have their canaliculi affected but not extensively enough to result in epiphora.

Antivirals are known to cause obstruction of the lacrimal drainage system, but the clinical picture is different. Idoxuridine was the first to be seen to affect the drainage system, and more recently trifluorothymidine (F3T) and adenine arabinoside (Ara-A) have been implicated. Such a complication of therapy is unusual, and it is the punctum that is seen to be occluded rather than the midzone of the canalicular system. Unlike the obstruction caused by herpes simplex virus, the punctal changes occurring with antiviral toxicity reverse on withdrawal of the drug.

The presence of midcanalicular occlusion is best established by passing lacrimal probes; macrodacryocystography is seldom satisfactory because of the difficulty encountered in achieving intubation. Usually the young patients affected with herpetic

Table 3  Causes of epiphora treated by canalicular surgery (1968–76)

<table>
<thead>
<tr>
<th>Cause</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral canaliculitis</td>
<td>26</td>
</tr>
<tr>
<td>Trauma</td>
<td>21</td>
</tr>
<tr>
<td>Congenital</td>
<td>15</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>11</td>
</tr>
<tr>
<td>Streptothrix</td>
<td>2</td>
</tr>
<tr>
<td>Stevens-Johnson syndrome</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>54</td>
</tr>
</tbody>
</table>
canalicular obstruction have severe watering, and surgery is often necessary. Failure to appreciate the nature of the postviral infection block can be compounded by inadequate surgery, the lateral extent of the lesion being overlooked. Dacryocystorhinostomy always fails in this situation.

Bypass surgery is necessary and a very successful treatment for this form of lacrimal drainage obstruction. The disadvantages of Lester Jones tubes are made light of by the young, highly motivated patients affected with postherpetic canalicular obstruction.

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References


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