Herpes simplex canalicular obstruction

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The characteristics of herpes simplex (herpes virus hominis) infection of the eyelids, conjunctiva, and cornea have been well documented. This case of canalicular obstruction following primary herpes simplex infection appears to be only the second recorded in the literature, and the first in which the diagnosis was confirmed by serological studies.

Case report

A 16-year-old girl was admitted to Bristol Eye Hospital in May, 1968. For the previous 3 days there had been a painful swelling of the right eyelids associated with a vesicular eruption of the margin of the right upper lid, and shortly before admission some vesicles appeared at the angle of the mouth. The girl's boy-friend had noticed a "cold sore" on his lip 11 days previously. The patient had never experienced any epiphora or excessive watering of the eye before this illness.

Examination

The oral temperature was 101°F. and there was bilateral enlargement of the preauricular, submandibular, and jugular lymph nodes. The right eyelids were oedematous and inflamed and there was ulceration and some vesicles on the margin of the upper lid. The oedema was sufficient to close the palpebral fissure. When the lids were separated, the conjunctiva appeared normal apart from some hyperaemia. The cornea was also normal and there was no staining with fluorescein. There were vesicles at the right angle of the mouth, on the dorsum of the tongue, and on the hard palate, with inflammation of the anterior gingival tissue.

Haemoglobin and white cell count were normal. Erythrocyte sedimentation rate (Wintrobe) was 29 mm. in the 1st hr.

A swab from the mouth yielded a scanty growth of Streptococcus viridans, diphtheroid sp., and Neisseria sp. Conjunctival swabs from both eyes showed a scanty growth of Staphylococcus aureus. Further discrete vesicles appeared on the right cheek and right upper lid shortly after admission at which stage the clinical photograph shown in Fig. 1 (opposite) was taken.

Diagnosis

Primary herpes simplex infection with secondary bacterial contamination.

Treatment

5-ido-2'-deoxyuridine drops were instilled hourly into the affected eye and into the vesicles, and neomycin ointment was applied four times a day to both eyes. Erythromycin 250 mg. 6-hrly was also given. The pyrexia settled 3 days after admission. By the eighth day the patient was able to open her eye completely and the vesicles had almost disappeared. She was discharged from hospital 2 days later.

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Follow-up

When seen in the Out-patients Department 2 weeks later she complained of epiphora, and on syringing the lacrimal apparatus there was some regurgitation of fluid, but some also passed on into the nose. The epiphora persisted, however, and probing 4 months later showed that both canaliculi were completely occluded although the puncta were normal; 6 months after the original illness it was decided to attempt a silicone tube intubation of the canaliculi (Keith, 1968a), but as it was impossible to pass any instrument along either canaliculus the operation was performed as follows:

The lacrimal sac was opened and appeared to be normal, as did the opening into it of the common canaliculus. A pigtail probe was passed retrograde from the sac to the punctum by force but the entire length of both canaliculi was completely occluded (Fig. 2), and there was no evidence that the probe was passing along any anatomical pathway. Monofilament nylon threads were passed from each punctum to the lacrimal sac by threading them through the eye of the probe and from there the nylon threads were passed down the naso-lacrimal duct and a silicone tube intubation was subsequently performed as described by Keith.

Postoperatively the epiphora was somewhat improved, but when the silicone tube was removed 6 months later the watering increased again, and examination has shown that both canaliculi are still completely occluded. At present further reconstructive surgery or a neurectomy of the lacrimal gland is being considered. It is interesting to note that one year after the initial illness there was a further eruption of vesicles on the right upper eyelid, but in this instance there was rapid spontaneous recovery.
During and after the illness complement-fixation tests for antibodies to herpes simplex were performed, and the results (Fig. 3) confirmed the clinical diagnosis. Initially no antibodies could be detected, but a month after the onset of symptoms there was a high level of circulating antibodies which gradually subsided.

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\text{FIG. 3} \quad \text{Changing titre of herpes simplex antibodies during and after the disease}
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**Discussion**

The lacrimal canaliculus, owing to its narrow calibre, is prone to inflammatory obstruction. This is demonstrated by the risk of occlusion or stenosis after irradiation, and even a long-acting miotic drug (furmethide iodide) has been shown to cause canalicular stenosis (Shaffer and Ridgway, 1951). Isolated canalicular obstruction is a relatively common condition, but the aetiology of most cases remains unexplained (Dalgleish, 1967) and occlusion caused by infective conditions is stated to be relatively rare (Werb, 1966).

Mycotic infection has been reported as a cause of obstruction but the mechanism appears to be a blockage of the lumen by debris and fungi rather than a narrowing of the canaliculus itself, as is shown by the response of these cases to irrigation with antibiotics and curettage (Pine, Hardin, Turner, and Roberts, 1960). (One of the patients with actinomycosis of the canaliculus reported by Pine and Hardin also had a stricture of the lumen, but the patient had previously had an attack of smallpox.)

Bacterial canaliculitis has also been occasionally described. There have been a few case reports of canalicular obstruction caused by Vincent's organisms (a mixed infection of fusiform bacilli and spirochaetal organisms) (Burns, Macnie, Pfeiffer, and Locatcher-Khorazo, 1958; Huysmans, 1962), but all these patients responded to local antibiotics and curettage. There is an isolated report of a case of *Pseudomonas pyocyanea* infection of the canaliculus, but this also responded to similar measures (Awasthy and Agrawal, 1963). It thus appears that a genuine canalicular occlusion (i.e. one caused by narrowing of the lumen and not by blockage by inspissated debris) is not caused by mycotic or bacterial infection.

By contrast it has been shown that viral infections may cause canalicular occlusion, although for most viruses except trachoma the number of case reports is very few. Obstruction due to trachoma has frequently been described. A radiological survey showed that 18 per cent. of cases had narrowing of the canaliculi (Gall, 1961), and Charamis (1957) stated in a review that the incidence of canalicular stenosis in trachoma varied from 0.5 to 80 per cent. according to different reports and different places.
Bouzas (1965, 1960) reported two cases of obstruction after ophthalmic herpes zoster and four after vaccinal conjunctivitis. Werb (1969) mentioned two cases of previous smallpox and chickenpox causing canalicular obstruction. A search of the literature revealed only one previous case report of obstruction following herpes simplex infection (Bouzas, 1965), although Keith (1968b) mentioned one case without giving any details.

In the case reported here there was an unusually severe primary herpetic infection of the eyelids, face, and mouth, with no obvious clinical invasion of the cornea and conjunctiva, which was followed by gradual total obliteration of both canaliculi. The diagnosis apart from clinical evidence was confirmed by the changing level of antibodies to herpes simplex in the patient's serum.

It could be argued that the occlusion was not caused by the virus infection but by the secondary bacterial invasion with Staphylococcus aureus; but there was never any clinical evidence of bacterial conjunctivitis, i.e. no mucopurulent discharge, and also the organism was cultured bilaterally. In the absence of any evidence of canalicular occlusion following primary staphylococcal conjunctivitis, it is unreasonable to implicate this organism in this instance where it was only a secondary invader.

It could also be possible that the occlusion was caused by the treatment with 5-iodo-2'-deoxyuridine (IDU), as Patterson, Fox, Davies, Maguire, Sellers, Wright, Rice, Cobb, and Jones (1963) showed, in a series of 198 patients with dendritic ulcers treated with IDU, that occlusion of the puncta occurred twice. However, in both cases, the occlusion involved only the puncta and was temporary, unlike the occlusion in the present case. It is also possible that the occlusion reported by Patterson and others (1963) was caused not by the IDU but by the herpes simplex infection.

Although recent surveys have shown a decrease in the percentage of adults with neutralizing antibodies to herpes simplex (Southam, Colley, and Clarke, 1968), infection with this virus remains extremely widespread and recurrent attacks at the same site as the primary eruption are very common. It is possible that the case described here is just an isolated clinical manifestation of herpes simplex which was brought about by the severity of the infection, but it is also possible that it represents the tip of the iceberg of a much larger number of patients who have herpes simplex virus established in the lacrimal canalicular mucosa, eventually causing stenosis and obstruction from recurrent attacks of low-grade infection. This rather tenuous hypothesis could perhaps be proved or disproved by statistical comparison of herpes simplex antibody studies in patients with canalicular stenosis and a group of matched controls. The most significant proven cause of inflammatory canalicular obstruction appears to be virus infection; and in non-trachomatous areas, the herpes simplex virus, by nature of its widespread incidence and tendency to recurrent infections, could be a significant factor.

It is also worthy of note that, if one had been aware of the final outcome of this case at an earlier stage in its clinical course, the passage of a silicone tube at the first sign of narrowing of the canaliculi might have preserved their patency.

Summary

A case of lacrimal canalicular occlusion following a severe herpes simplex infection is described. Although this is only the second case reported in the literature, it is suggested that herpes simplex infection may be a significant cause of canalicular obstruction. The literature concerning other infective causes of such an obstruction is briefly reviewed.
I wish to record my thanks to Mr. C. G. Tulloh, under whose care the patient was admitted, and also to Dr. R. G. Smith of the Bristol Dental Hospital, who supplied the material for Fig. 3 and who has reported on oro-dental aspects of this case.

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