Congenital deficiency of meibomian glands

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SUMMARY A 16-year-old girl presented with contact lens intolerance. She was found to have a marked deficiency of meibomian glands in the upper lids and almost total absence in the lower lids. Evidence of tear film instability was found and attributed to deficient lid oil production. A daily wear soft contact lens was later fitted and tolerated.

Meibomian lipid forms the anterior layer of the tear film.¹ It is believed to retard evaporation from the precorneal tear film and to prevent contamination of the tear film by the more polar skin sebum of the cutaneous surface of the lid margin.

Meibomian lipid is secreted by the meibomian glands, which are radially disposed and serially arranged within the substance of the tarsal plate, with their ductular openings placed on the cutaneous side of the mucocutaneous junction. This ideal placement permits the delivery of meibomian oil on to the anterior aspect of the tear film, where it spreads over the aqueous film as the eyes open after each blink.

Histologically the glands are racemose holocrine glands, whose acini open into a central ductule lying at right angles to the lid margin. The acini form a series of yellowish grape-like clusters, distributed at intervals along the length of the central ductule and clearly visible through the conjunctiva of the tarsal plate in young adults. With increasing age these acinar details are less visible, though a yellow streak can be seen representing each gland. The glands are also visible by transillumination through the lid,^{2,5} and an interesting infrared technique (meibomioscopy) has been developed.

The ductular orifices at the lid margin are visibly surrounded by a cuff of epithelial tissue, which has a delicate vasculature visible by fluorescein angiography.³ The orifices are flush with the surface in health and arranged, usually in a single line, parallel but anterior to the mucocutaneous junction. At times the line is less regular and may give the impression of a duplication in the antero-posterior direction. The epithelial cuff is of similar pink colour to neighbouring lid margin tissue. In black people the orifices are outlined by pigment, and are more readily seen.

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There are more glands in the upper than the lower lid (30–40 in the upper and 20–30 in the lower). Gentle pressure through the lid on to the globe impresses the tarsal glands and expresses a clear fluid lipid on to the lid margin. Meibomian oil is liquid at lid temperature (with an upper melting point range at 32.0–33.8°C).⁶ The stimulus for secretion of meibomian lipid is not known.

Although disease of the meibomian glands is common as part of meibomitis, in association with marginal blepharitis, or with chalazia, or rosacea, there are few reports relating such abnormality to surface ocular disease. However, various authors^{7–10} referred to conjunctivitis accompanying meibomian gland disease, and Bron and Tripathi (1973) to a specific form of epithelial keratopathy caused by meibomianitis (cystic epithelial keratopathy).¹¹ Thygeson¹² considered the role of staphylococcal toxins in blepharoconjunctivitis. Epithelial keratitis has been described by McCulley and Sciallis associated with meibomianitis.¹³ It has been assumed that such manifestations are due to a deficiency of the normal conservative function of the meibomian lipid in retarding evaporation, and represent an induced non-wetting eye disease.

An alternative explanation relates to bacterial infections of the meibomian glands and to suggested toxic meibomian lipid degradation products.^{13,14} The action of fatty acids released by bacterial lipases is a related hypothesis.¹⁵

Case report

The present paper reports absence of meibomian glands in a young contact lens wearer. In September 1981 a 16-year-old white American girl (OEH 196501) was referred to the Oxford Eye Hospital

Fig. 1 Diagram to illustrate the disposition of residual meibomian glands.

from an American Air Base with a history of one and a half months of soft contact lens wear using cold chemical sterilisation. She reported increasing redness of the eyes, and in the two weeks before presentation she developed marked pain in both eyes. She stopped lens wear for one week and was started on chloromycetin solution qds without relief. A marked 'corneal stipple', with punctate staining in the left more than the right eye, failed to resolve over this period. Acuity was reduced in each eye.

Acuity in the casualty department was RE 6/9 and LE 6/12 corrected, improving to RE 6/5 and LE 6/6+3 with a pinhole. There was bilateral punctate keratopathy. Examination in the external disease clinic two days later showed bilateral upper tarsal conjunctival papillae of mild degree and a few follicles and cystic changes below. There was a 2+ interpalpebral conjunctival and corneal punctate epithelial staining. Anterior segments and fundi were otherwise normal.

At the next visit it was recognised that there was a major abnormality of the meibomian glands which were noted to be both deficient in number and abnormal in morphology (Fig. 1). The right upper lid had 14 orifices associated with obliquely disposed glands about a quarter their normal length. They were absent from the central part of the lid. In this region there was the occasional suggestion of a gland which failed to come in contact with the surface of the lid margin. The right lower lid showed a single gland and orifice. On the left the upper lid had 16 orifices. Glands were elongated and obliquely disposed in the temporal part of the tarsal plate, and more vertically disposed in the nasal part of the lid. Many were approximately of normal length. The left lower lid had a single short gland about half the normal length. On either side of this solitary gland was a line of dark, rounded spots in the line of the subtarsal fold, which were assumed to be abortive tarsal gland rudiments. There was no connection with the lid margin. Just temporal to the solitary gland orifice was

a patch of presumed squamous metaplasia across the breadth of the lid margin.

Otherwise the lid margins were normal in appearance, and, where meibomian orifices were visible, they were disposed at their normal location in the mucocutaneous junction; slight pouting was present in some. Oil was expressible from the upper lid gland orifices on the right. No oil was expressible from the other lids. Over the period December 1980 to 1981 the fluorescein breakup time (BUT) was measured on a number of occasions: R 4–8 s, L 6–8 s; R 15 s, L 5 s; R 9 s, L 7 s; R 5 s, L 6 s. Schirmer's test was R 15 and 12 mm in 5 minutes, and L 10 and 5 mm on separate occasions.

The patient was a myope. Refraction was R $-2.75/+0.5$ axis $25^\circ=6/4$ and L $-2.75/+0.50$ axis $170^\circ=6/4$. K readings were R 7.5 along 90° ; 7.35 along 150° and L 7.5 along 110° ; 7.40 along 30° . About one year after presentation she was refitted with a Scan soft lens and achieved all day wear (7:00 am–9:00 pm in February 1982). There was a tendency to stinging in the left eye during wear, after prolonged staring. There was a tendency to accumulate deposits on the right lens, which necessitated changing the wearing schedule.

Discussion

The present study reports a patient with meibomian glands which are deficient in number, rudimentary in form, and abnormally disposed. The absence of any but minor conjunctival signs (interpalpebral staining and upper tarsal papillae) and the youth of the patient strongly suggest that this is a congenital abnormality of the glands. The patient was otherwise normal, and there was no skin complaint. There are few reports of congenital absence of meibomian glands. Holly and Lemp refer to an association with anhydrotic ectodermal dysplasia.¹⁶

One of us (AJB) has seen a similar case, also female, in another institution. In this patient

symptoms were present from infancy.

Symptoms in our patient are assumed to be due to a deficiency of meibomian oil. Meibomian oil is thought to prevent evaporation from the ocular surface. In the rabbit Mishima and Maurice estimated that meibomian oil reduced evaporation by 4–20 fold.^{17,18} Brown and Dervichian¹⁹ could not confirm this conservational effect of meibomian oil on water loss. But, although human meibomian oil has a different composition from rabbit meibomian oil,^{20,21} it is difficult to accept that it does not perform this function, since it forms a multimolecular sheet across the preocular surface between blinks in normal subjects. The other postulated function of meibomian oil is to prevent contamination of the ocular surface with sebaceous lipid present on the lid skin²² and derived from lash-associated sebaceous glands. The ability of sebum to lower the surface tension of preocular tear meibomian lipid has been graphically demonstrated by McDonald, who showed that a sebum covered hair touched on to the preocular surface causes an immediate disruption of the lipid film.²³ The presence of hydrocarbons in meibomian oil collected by expression from normal lids^{21,24} suggests that some contamination of the meibomian secretion may occur normally. Norn has shown that lipid on the lid margin can find its way into the conjunctival sac and draws the same conclusion.²⁵

A deficiency of normal lid oil in the tear film and perhaps the presence of sebum on the tear film would be expected to encourage evaporation from the ocular surface and symptoms and signs of non-wetting. The interpalpebral staining pattern and reduced BUT recorded in our patient are in keeping with this. In the study by Bron and Tripathi, a particular form of epithelial surface change was observed and termed cystic epithelial keratopathy, associated with the meibomian blockade and other features of meibomianitis.¹¹ In that situation epithelial cysts are observed in the exposed region of the cornea, together with punctate erosions and punctate epithelial keratopathy.

In our patient symptoms appeared after institution of contact lens wear, despite the likelihood that the oil gland deficiency was congenital. This suggested that contact lens wear precipitated the surface changes. Punctate interpalpebral staining gradually decreased once contact lens wear had been stopped but did not entirely disappear. Punctate corneal changes may be slow to subside after contact lens problems whatever their cause; therefore it is not certain that their persistence was due to the poor wetting or to contact lens 'insult'. It is our impression that clinical presentation of non-wetting problems due to aqueous deficiency or meibomianitis of congenital or early onset does not necessarily occur in

infancy but may start to produce symptoms in the teens or twenties. This suggests that the ocular surface may be more resilient in the young. A change in lens and regimen ultimately permitted contact lens wear in this patient.

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