

LETTERS TO THE EDITOR

Dear Editor,

Periorbital Allergic Contact Dermatitis Caused by Lanolin in a Lubricating Eye Ointment

Periorbital dermatitis encompasses a group of inflammatory dermatoses. Allergic contact dermatitis (ACD) is usually considered the most common cause, but differentials include irritant contact dermatitis, atopic dermatitis, seborrhoeic dermatitis, rosacea, psoriasis, infections and infestations.¹ Periorbital ACD caused by topical ophthalmic agents has been well described.^{2,5} Principal allergens include preservatives, anti-microbials and anti-glaucoma agents. We describe the first case of periorbital ACD caused by lanolin in an over-the-counter ocular medicament.

A 52-year-old atopic man presented with a 12-month history of sore, irritated eyes, together with eyelid erythema and patchy facial dermatitis. He had previously undergone five corrective procedures for bilateral floppy eyelid syndrome. As a result of persistent eyelid mal-opposition, the patient required twice-daily ocular lubricants to prevent corneal damage. For 18 months his regimen had comprised GenTeal Gel (Novartis, Sydney, NSW, Australia) twice daily, Hylo-Forte Eye Drops (Scope Ophthalmics, UK) mane and Refresh Lacri-Lube Lubricant Eye Ointment (Allergan, Sydney) nocte. Following the development of periorbital symptoms he had trialled oral corticosteroids and pimecrolimus cream with short-lived improvement. Fluoromethalone eye drops had helped, but were ceased due to increased intra-ocular pressures. His comorbidities included psoriasis, hypertension and obstructive sleep apnoea requiring continuous positive airway pressure.

Patch-testing was performed according to the International Contact Dermatitis Research Group criteria with the Australian Baseline Series, common ocular allergens and the patient's own samples. He developed positive reactions at days 2 and 4 to wool alcohols (50% pet.) and Amerchol L-101 (50% pet.) (Chemotechnique, Vellinge, Sweden), both lanolin derivatives. There were no other positive reactions. The patient's only source of exposure to lanolin was the Lacri-Lube eye ointment, which contained wool alcohols 0.2% w/w. The other ingredients were liquid paraffin 42.5% w/w and soft white paraffin 57.3% w/w. The ointment was preservative-free.

The suspected causative agent, Lacri-Lube eye ointment, was promptly ceased. He continued using the remaining two ocular lubricants. The patient's periorbital and facial

dermatitis had completely resolved several weeks later. Six months later he remained symptom free. A repeat open application test (ROAT) performed for 3 weeks with Lacri-Lube ointment was negative.

DISCUSSION

Lanolin derivatives are used extensively for their emollient qualities in topical therapeutics, personal care items and cosmetics.⁴ Recent studies suggest that the prevalence of ACD to lanolin is low in healthy individuals.⁴ Lanolin is a weak sensitizer in normal skin but may easily sensitize damaged skin.⁴ Cases of ACD to lanolin in cosmetics and topical medicaments, but not ophthalmic agents, have been reported.^{5,6}

Ours is the first clearly defined case of lanolin in an ophthalmological medication causing periorbital ACD. The literature includes several large cohorts of patients with periorbital ACD, where lanolin derivatives are included in a long list of contact allergens.^{1,2,7,8} However, these epidemiological studies do not list the source of lanolin exposure for individual cases. Thus, it is possible these patients reacted to lanolin from cosmetics, in which it is a relatively common ingredient.

Although our patient did not react to Lacri-Lube tested 'as is' during patch-testing or the subsequent ROAT, it is recognised that the low concentration of allergens in ophthalmologicals may be insufficient to elicit an allergic reaction on thick healthy skin.⁹ In contrast, the thin eyelid skin presents a lower threshold for elicitation, particularly if it is already damaged. In our patient, skin barrier disruption from recurrent eyelid eversion and multiple surgeries, related to his floppy eyelid syndrome, was likely to have contributed to the development of his lanolin allergy. The ointment may also have caused his facial dermatitis through contact with his hands or pillow.

Lanolin-containing ocular lubricants present a rare but important source of periorbital ACD. Although present in very low concentrations, lanolin in such medicaments may be sufficient to cause ACD on thin eyelid skin. At-risk patients may include those with pre-existing eyelid skin disruption. Our case alerts dermatologists and ophthalmologists to a potential new cutaneous allergen source.

Abbreviations:

ACD	allergic contact dermatitis
ROAT	repeat open application test

Conflict of interest: none

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Dear Editor,

Adopting the orphan: The importance of recognising hidradenitis suppurativa as a systemic auto-inflammatory disease

Hidradenitis suppurativa (HS) has been referred to as the orphan disease¹ of dermatology¹ as there is little understanding of its pathogenesis or of effective options for therapy. Translational biology has provided us with evidence to suggest that HS is a systemic auto-inflammatory disease and is not just confined to the skin.² The advent of monoclonal antibodies has provided a new treatment paradigm for various inflammatory disorders and HS is no exception.² Understanding HS as a systemic disorder with comorbidities that contribute to disease activity is essential if dermatologists are to effectively manage this burdensome disease.⁵

HS is a chronic inflammatory condition presenting with deep-seated nodules and abscesses, commonly in the inguinal, axillary and sub-mammary regions. HS is largely

resistant to current medical therapies and exerts a large impact upon the patients' quality of life.¹ Individuals with HS have recurrent episodes of painful, discharging sinuses and often end up in the hands of surgeons. They undergo large excisions of apocrine-bearing skin and subcutaneous tissue with the resultant cosmetic and functional impairment that surgery and its complications can entail.

The advent of biological therapies and the off-label use of adalimumab and infliximab for HS have given new hope for effective systemic therapy for this disorder.² The emergence of these new therapies has coincided with new insights into the pathogenesis of HS, revealing commonalities between HS and auto-inflammatory diseases including familial Mediterranean fever and Muckle-Wells syndrome.⁴ Auto-inflammatory diseases are characterised by periodic or intermittent fevers and severe, localised inflammation in the absence of infectious or autoimmune aetiology.⁵ Recent expanded definitions of HS include conditions without fever and with persistent inflammation and dysregulation of the innate immune system.⁵ The commonality between all these conditions is the dysregulation of the inflammasome.⁵ The inflammasome is a disparate collection of cytoplasmic biochemical pathways and the common end pathway for various inflammatory conditions including gout, diabetes, psoriasis and atopic dermatitis.⁵ Dysregulation of the inflammasome has been identified in HS associated with inherited polymorphisms in gamma secretase and Notch-associated proteins.⁵ These proteins are affected by hormones including oestrogen and progesterone, smoking and adipokines associated with insulin resistance and obesity; all factors that are known to be exacerbants of HS. The downstream activation of the inflammasome results in elevated levels of interleukin 1B, elevated tumour necrosis factor (TNF)- α , Th17 immune response, deficient follicular support and follicular occlusion, resulting in the clinical manifestations of HS.⁴

The clinical importance of considering HS as an auto-inflammatory disease includes the necessary recognition of systemic comorbidities, including visceral adiposity, insulin resistance and hormonal factors that contribute to the activation or exacerbation of the disease. An increased understanding of the pathogenic pathways involved (Fig. 1)⁵ and the way these are correlated with differential clinical phenotypes may also allow targeted therapies to specific patient subgroups.

The use of TNF- α inhibitors with adjuvant therapies for contributing comorbidities such as metformin for insulin resistance and aggressive weight loss strategies give hope for the effective management of HS.^{1,3,4} We hope that further research into the phenotypic variations in HS presentations will lead to more

Abbreviations::

HS	hidradenitis suppurativa
TNF	tumour necrosis factor

Conflict of interest: none