of cutting oils, suds, lubricating oils and greases. Machinists' and fitters' hands are always contaminated by grime, which contains oil and swarf; tools they grip and rotate are similarly contaminated. Hands so contaminated are wiped on contaminated rags. Not one worker can escape microtrauma and yet the incidence of dermatitis is relatively low. It may well be that work trauma affects the skin but the question which has to be answered in court is: is it negligent to expose men to work trauma?

It is necessary to show that there is a risk to reasonably normal skin, and a small incidence of dermatitis in like circumstances in industry generally augers against there being a risk against which an employer can be expected to guard, particularly when the precautions which would be suggested, namely gloves, involve dangers both in context of their use with machinery and from sweating.

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Melkersson's Syndrome

R E Bowers MD FRCP (Gloucestershire Royal Hospital, Gloucester, GL1 3NN)

Mrs E B, aged 45

History: In 1944 she suffered a left facial palsy which cleared completely. In 1949 she was thought to have geniculate herpes with another attack of left facial palsy which did not recover. Subsequently she had recurrent attacks of pain, swelling of the face and cervical adenopathy, which ceased only during her three pregnancies.

At the end of 1964 she was delivered of her third and last child. Within three months she had three attacks of cellulitis of the face, in the third of which I saw her for the first time and found: residual maculopapular eruption of right cheek and left chin resembling a healing herpes simplex;

cranial nerves normal apart from almost total paralysis of left facial nerve; scrotal tongue.

Between 1964 and 1970 she has had eleven attacks of cellulitis of the face, often with constitutional symptoms. In December 1967 she had a particularly severe attack beginning with sublingual swelling, and developed a right facial palsy which has not recovered.

Investigations: 18.2.65: Herpes simplex complement-fixing antibody titre less than 1:5 (shortly after attack). 11.8.70 and 21.2.72: herpes simplex CFT 1:8 (during attack). 2.3.72: herpes simplex CFT 1:8 (after attack). 13.3.72: total protein 6.6, albumin 4.0, α_1 -globulin 0.3, α_2 -globulin 0.5, β -globulin 0.8, γ -globulin 1.0 g/100 ml. Immunoglobulins: IgG 932, IgA 103, IgM 83 mg/100 ml (normal 513–1615, 112–372, 47–170 mg/100 ml).

1968: Negative Kveim test and normal delayed type hypersensitivity (Dr G James).

November 1970 and January 1972: Herpes simplex virus isolated (Dr E Wright, Gloucester Public Health Laboratory) from virus cultures of early lesions made at Professor C D Calnan's suggestion.

Treatment: The following drugs were used with no apparent effect: tetracycline, phenoxymethyl penicillin, intralesional triamcinolone (even though injected forty-eight hours after onset of an attack), ACTH, tetracycline with ACTH, chloroquine, prednisolone 21-stearoylglycolate, norethisterone, compresses of idoxuridine in dimethyl sulphoxide, and systemic cytarabine.

Comment

This woman had never developed herpes simplex antibodies in spite of attacks of inflammation of the face which are associated with proven herpes simplex virus infection. Is it possible that Melkersson's syndrome results from impaired production of herpes simplex antibodies? Perhaps the scrotal tongue is a genetic marker of some immunological abnormality.

Chronic Leg Ulceration ?Connective Tissue Defect Julia P Ellis MB MRCP

(Department of Dermatology, United Oxford Hospitals, Oxford)

Miss BS, aged 29

History: Persistent widespread ulceration of both legs since 1960. The ulcers appear without preceding trauma but occur mainly at pressure areas on the backs of her thighs and round her ankles. She was investigated by Dr I Anderson and Dr I B Sneddon in 1961 who suggested the diagnosis of Ehlers-Danlos syndrome. In 1967 treatment with i.v. low molecular weight dextran