Stucco keratoses are benign acquired papular warty lesions which usually occur on the distal parts of the lower limbs of elderly men. Their nature and pathogenesis are uncertain. Eight patients with multiple stucco keratoses who presented to the Cardiff Dermatology Department over an 18-month period were studied in an attempt to characterize these lesions. All subjects were male, with ages ranging from 50 to 72 years. Their lesions were most numerous over the fronts of the lower legs, but were also evident over other areas of the limbs. Most patients had a history of prolonged solar exposure, but the relationship of this to the development of the lesions was uncertain. Histologically there were no dysplastic changes and a characteristic regular ‘church spire’ type retention hyperkeratosis without marked acanthosis was evident. Lack of a marked increase in the epidermal cell population size is reflected in the normal in vitro tritiated thymidine autoradiographic labelling indices (7.81 ± 1.67%, normal range 5 to 9%). Ultrastructurally no viral particles were evident and a normal pattern of epidermal differentiation with hypergranulosis was present. Two of the patients with myriads of lesions who were treated for 2 months with cetacinate showed a dramatic clinical improvement which, however, lasted for 6 months only.

(accepted October 1, 1990.)


R. Marks, Department of Dermatology, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN, Glamorgan, Wales, Great Britain.

Stucco keratoses are benign acral papular warty lesions usually occurring on the distal parts of the lower limbs of elderly men. The name was first used in Australia in 1958 by Kocsard et al. to describe lesions seen in 24 male patients while carrying out a survey on 250 geriatric subjects (1). The name ‘stucco’ stems from the ‘stuck on’ appearance of the lesion on the skin. The nature of these odd keratotic lesions is uncertain and nothing is known concerning their cause. We have studied 8 patients with the condition and in this paper describe our attempts at characterizing stucco keratoses from both the structural and pathophysiological standpoints.

PATIENTS, MATERIALS AND METHODS

Clinical details

Eight patients attending the University of Wales Hospital Dermatology Outpatient Department between July 1985 and January 1987 were diagnosed clinically as having stucco keratoses. For the purpose of this study, stucco keratoses were defined as multiple non-pigmented hyperkeratotic warty lesions up to 1 cm in diameter, affecting any surface of the lower legs and to a lesser extent the thighs, forearms and upper arms. The lesions did not possess the clinical features of viral warts, seborrheic warts or solar keratoses. Full clinical details of the patients studied are given in Table I. They all gave their written, witnessed informed consent for inclusion in this study.

Materials and methods

Biopsies of representative lesions were divided into four segments by scalpel and one portion fixed in 10% formalin, dehydrated and embedded in paraffin wax, then sectioned and stained with haematoxylin and eosin for light microscopy.

Parts of the biopsies were also quenched in hexane and cryostat sections were cut on a Bright’s motorized cryostat and examined for deposits of IgG, IgM, IgA, C3 and fibrin by standard direct immunofluorescent techniques using appropriate controls (2).

Cryostat sections were also examined histochemically for non-specific esterase, glucose-6-phosphate dehydrogenase, succinic dehydrogenase and dopa oxidase enzyme activities. Non-specific esterase activity was detected by the indoxyl acetate method (3). Glucose-6-phosphate dehydrogenase, succinic dehydrogenase and dopa oxidase activities were detected according to the methods described by Chayen et al. (3).

Portions of lesions were divided into 1–2 mm thick slices, incubated for 4 h at 37°C in Eagle’s MEM containing tritiated thymidine (18 Ci/mmol) and subsequently fixed, processed, wax-embedded and then developed autoradiographically using the dipping film method (4). Small fragments of all lesions were also prepared for transmission electron microscopy by initial fixation in Karnovsky’s fixative, secondary fixation in osmium tetroxide and embedding in Araldite prior to sectioning and staining with uranyl acetate and lead citrate. During inspection of the EM sections, special care was taken to determine the presence of viral particles. Viral cultures were also attempted from

Acta Derm Venereol (Stockh) 71
Table 1. Details of patients studied

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Reason for consultation</th>
<th>Skin type</th>
<th>Site of stucco keratoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>Lesion right side of neck, proven squamous cell carcinoma</td>
<td>2</td>
<td>Hands and feet</td>
</tr>
<tr>
<td>2</td>
<td>63</td>
<td>Progressive systemic sclerosis</td>
<td>3</td>
<td>Feet</td>
</tr>
<tr>
<td>3</td>
<td>72</td>
<td>Psoriasis and solar keratoses</td>
<td>2</td>
<td>Feet</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>Renal transplant with numerous warty lesions</td>
<td>2</td>
<td>Arms and legs</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>Numerous warty lesions, proven stucco keratoses</td>
<td>2</td>
<td>All 4 limbs</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>Solar keratosis</td>
<td>2</td>
<td>Feet</td>
</tr>
<tr>
<td>7</td>
<td>64</td>
<td>Numerous warty lesions, proven stucco keratosis</td>
<td>2</td>
<td>Whole body, mainly extremities</td>
</tr>
<tr>
<td>8</td>
<td>56</td>
<td>Renal transplant with warty lesions</td>
<td>3</td>
<td>Legs</td>
</tr>
</tbody>
</table>

fragments of skin by the Virus Laboratories of the Public Health Laboratory Service.

RESULTS

Clinical findings

The numbers of lesions present ranged from 5 to more than 250. The anterior aspect of the lower leg just above the ankle, the ankle and the dorsum of the foot were the usual sites of the lesions. They also occurred on the posterior aspect of the forearms (Fig. 1) and in the patients with the highest number of lesions the trunk was involved. The keratoses were irregularly shaped, flat topped, grey dusky or pink in colour, rough to touch, and measured 1–4 mm in diameter. All the lesions left a collarette of scale without bleeding or exudation of the exposed skin when the scale was dislodged.

It was difficult to quantify the total amount of solar exposure to which each patient had been exposed. Half the patients had worked out of doors or their leisure time was spent outdoors. All had been abroad on holiday but only 2 had worked in tropical climates. Seven patients had clinically evident photo-aging, 3 had concurrent facial solar keratoses, 1 also had a squamous cell carcinoma and one had a lentigo maligna affecting the face. Two patients, numbers 5 and 7, troubled by the most numerous lesions, were treated with etretinate, initially 1 mg/kg/day for 1 month then 0.5 mg/kg/day for a further month. There was a dramatic improvement and both patients were free of lesions 6 weeks after starting the treatment. However, 6 months after cessation of treatment the lesions recurred. One patient requested further treatment and after one month of 1 mg/kg/day etretinate is now on a maintenance dose of 25 mg etretinate on alternate days and remains lesion free.

Investigative findings

Histologically the epidermis assumed a regularly peaked, 'warty' configuration, the so-called 'church steeple' hyperkeratosis and some acanthosis.

Fig. 1. There are numerous stucco keratoses on the arm of this patient.

Fig. 2. Photomicrograph of stucco keratosis showing 'church steeple' hyperkeratosis and some acanthosis. H & E, x150.
steeple' hyperkeratosis (Fig. 2). There was only moderate overall acanthosis, the granular cell and prickle cell layers were somewhat thickened but normal and there was no atypia of the epidermal cells. No horn cysts or nests of basaloid cells were present. The dermis was normal and not inflamed. Overall the lesions most resembled acanthosis nigricans or epidermal naevi.

Direct immunofluorescence studies of cryostat sections for IgG, IgM, IgA, C3 and fibrin were negative. Histochmical analysis of frozen sections for non-specific esterase activity, which is normally confined to the granular layer, showed denser and broader deposition of indigo reaction product than would normally be expected. Glucose-6-phosphate dehydrogenase activity, usually confined to the granular layer, showed a small increase in activity as depicted by the increased deposition of formazan reaction product in the granular cell layer. Succinic dehydrogenase activity, usually uniform throughout the epidermis, showed a slight decrease in activity as seen by the lesser deposition of the reaction product than would normally be expected. Dopa-oxidase activity showed a normal distribution of melanocytes along the basal layer. The normal in vitro tritiated thymidine autoradiographic labelling indices (6.37% to 8.72%, mean 7.81% SD 1.67%, normal range 5 to 9%) suggest that the rate of epidermal cell production is unchanged in these lesions.

Ultrastructurally, a normal pattern of differentiation was evident in the epidermis of lesions examined by transmission electron microscopy. The presence of viral particles was not detected. Viral cultures of skin were also negative.

DISCUSSION
In this study we attempted to characterize the nature of stucco keratoses by examining their clinical and pathological features in detail. We were surprised at being able to identify 8 patients over 18 months. In fact stucco keratoses are probably more common than previously thought. The reason for this may be that the lesions are usually inconspicuous and asymptomatic and therefore not brought to the attention of the physician. Green, when surveying 200 non-dermatological patients in Australia, found an incidence of 20% (8). A similar but smaller study he undertook in England revealed an incidence of 14%. All our patients were male, which is consistent with other reports that this disorder is more prevalent in males (8). The two youngest had had renal transplants and were receiving immunosuppressive therapy. Such patients are at a greater risk of developing dysplastic lesions and viral warts (5), and although the stucco lesions were neither of these former lesions it cannot be denied that the patients' underlying disorder and/or its treatment may have predisposed to the development of stucco keratoses in relatively young patients.

We do not know what role solar damage plays in the pathogenesis of this disorder. Some authors have proffered solar exposure as a cause (9), others have been more in favour of exposure to heat and chemicals, especially tar (9). All the patients in our study had had considerable sun exposure and demonstrated evidence of photo ageing. The stucco keratoses, however, were not confined to sun-exposed sites and were not particularly prevalent on the most sun-damaged areas. None of the patients in this study had been exposed to tar. Histologically there was no evidence of dysplasia. The major feature on light microscopy was the presence of 'church spire' hyperkeratosis and slight regular acanthosis. They have been regarded as variants of seborrhoeic warts (10) and it may well be that this is the case, despite their unusual distribution. The histochemical results showing a slight increase in non-specific esterase and glucose-6-phosphate dehydrogenase activities as well as a slight decrease in succinic dehydrogenase activity are common in conditions characterized by hypergranulosis and ortho-hyperkeratosis. These investigative findings suggest that the disorder represents an acquired focal abnormality of keratinization in which aspects of the differentiation process are enhanced.

In conclusion, we believe that stucco keratoses may be more prevalent than previously thought, that although they appear to arise in individuals with concurrent solar damage, they themselves are benign in nature, that they represent an acquired focal abnormality of keratinization and that they respond to treatment with etretinate.

REFERENCES
Exzema craquelé can be induced by repeated open application of a topical glucocorticoid, viz. 0.05% clobetasol 17-propionate cream. This might not be invariably due to the active component. Comparison of the skin surface roughness as assessed by profilometry and as expressed by R\textsubscript{2DN} showed a decrease after repeated open application of 0.1% betamethasone 17-valerate cream and 0.25% prednicarbate cream, but an increase following the vehicle of the latter preparation. Thus commercial oil-in-water emulsion preparations seem to be potentially injurious to human skin, though this may be masked when a glucocorticoid is added.

(Accepted October 16, 1990.)

H. C. Korting, Department of Dermatology, Ludwig-Maximilians-Universität, Frauenlobstr. 9–11, D–8000 München 2, FRG.

As early as in 1979 Kligman & Frosch (1) described a clinical picture encountered after discontinuing prolonged application of topical glucocorticoids, characterized by reddening, tenderness, itch, scratching and scaling. While Kligman & Frosch traced these findings to what they called steroid addiction it has subsequently been described how regular application of topical glucocorticoid preparations can even in a very limited period of time lead to a diseased state presenting as eczema craquelé (erythema craquelé) (2). According to Björnberg (2) this is especially linked to repeated application of potent glucocorticoids in a cream base, such as 0.1% betamethasone 17-valerate or 0.1% hydrocortisone 17-butyrate, when applied under occlusion. Yet in rare cases it may also be due to the open application of a potent steroid cream (over a 6-week period) or to the repeated application of pH 5 ethanol base under occlusion. Eczema craquelé is associated with skin dryness. Skin dryness defined as a clinical condition meaning a rough, finally scaling non-inflamed skin surface can adequately be assessed by using surface profilometry (3). When repeatedly confronted with eczema craquelé as a side effect of the repeated open application of clobetasol 17-propionate cream (0.05%) during a trial performed for the comparative assessment of skin atrophy due to various topical glucocorticoids, we looked for skin surface roughness before and after the application period in a subgroup of 6 healthy volunteers.

MATERIAL AND METHODS
During a double-blind randomized trial, four different formulations were applied to the proximal flexor part of the forearms of 24 healthy volunteers. Application followed a randomized blocks design to ensure that either preparation A or B was applied to the one forearm and preparation C or D to the other. Preparation A representing 0.25% prednicarbate cream, B the corresponding vehicle; C, 0.1% betamethasone 17-valerate cream; C, 0.05% clobetasol