Table I. The growth of Pitysporum orbiculare from scrapings from patients with tinea versicolor

<table>
<thead>
<tr>
<th>Sabouraud agar with addition of</th>
<th>Growth of Pitysporum orbiculare</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sun lotion</td>
<td>2</td>
</tr>
<tr>
<td>Cocoa butter</td>
<td>5</td>
</tr>
<tr>
<td>Sesame oil</td>
<td>5</td>
</tr>
<tr>
<td>Eight other ingredients of the sun lotion*</td>
<td>0 10</td>
</tr>
<tr>
<td>Coconut oil</td>
<td>8</td>
</tr>
</tbody>
</table>

* Ethylene-diglycol, liquid paraffin, glycercamino- benzoate, Comperlan KD (mixture of ethanalamides of coconut acid), Butylhydroxy anisole, Triethanol amine, propaloid T., stearin.

A man, 26 years, had had constant recurrence of tinea versicolor since 1973—always localized to the abdomen only, and always recurring after treatment. His wife had ichthyosis and used cream every day—and she also proved to have tiny spots of tinea versicolor.

In some cases application of creams also explains the occurrence of tinea versicolor in areas with few sebaceous glands, such as the elbow flexures and genital folds.

The purpose of this paper has not been to present new data related to the growth of *Pitysporum orbiculare* on different lipid constituents of body lotions, but to call clinicians' attention to a possible relationship between recurrent tinea versicolor and the use of body lotions.

The problem could be solved by the use of vaseline or lotions not containing fatty acids, which stimulate the growth of *Pitysporum orbiculare* (7).

ACKNOWLEDGEMENT

My thanks are expressed to B. Sylvest, M.D., for help with the microscopical examination of the cultures.

REFERENCES


Nail-Patella Syndrome Associated with Renal Failure Requiring Transplantation

Jesper Verdich

Department of Dermatology, Odense University Hospital.
DK-5000 Odense, Denmark

Received March 19, 1980

Abstract. The nail-patella syndrome is a rare, inheritable disease of the connective tissue in which multiple osseous abnormalities are associated with dysplasia of the nails. Renal dysfunction is found in 25-42% of cases and varies from asymptomatic proteinuria to renal failure. This paper describes a patient with nail-patella syndrome and terminal renal disease requiring transplantation.

Key words: Nail-patella syndrome; Terminal renal disease; Renal transplantation

The nail-patella syndrome or hereditary osteoonycho-dysplasia is a rare, inheritable, autosomal dominant disease of the connective tissue. The principal manifestations include nail dysplasia, skeletal deformities involving the elbow joint, iliac horns and hypoplasia or a plasia of the patellae. Renal dysfunction has been found in 25-42% of reported cases (2, 9) and most frequently presents as asymptomatic proteinuria. However, terminal renal disease has also been reported (3, 8, 10).

This paper presents a patient with nail-patella syndrome associated with renal failure requiring transplantation.
CASE REPORT

The patient, a 15-year-old girl, had for the first time moderate proteinuria at age seven. An intravenous urography performed then showed normal pyelograms except for a slight dilatation of the calyces. Renal function was normal, as measured by serum creatinine and creatinine clearance. During the following 2 years, the renal function was unchanged, with persistent proteinuria. At the age of nine she was admitted to a psychiatric hospital because of behavioural disorder. Clinical examination showed an increased carrying angle of the elbows with extension limited by 15 degrees and supination by 25 degrees. The patellae were hypoplastic and the nails showed marked dysplasia. Laboratory studies were normal apart from moderate proteinuria. On the basis of these findings the diagnosis nail-patella syndrome was suggested.

Following discharge from the psychiatric department at the age of ten she was subsequently lost to follow-up but apparently did well until the age of fourteen when she was admitted to a hospital because of hypertension and severe impairment of renal function. On admission, blood pressure was 200/140 mmHg, serum urea nitrogen 43.2 mmol/l (normal range 3.0-7.8 mmol/l), serum creatinine 946 µmol/l (normal range 43-135 µmol/l) and haemoglobin 4.6 mmol/l (normal range 6.9-9.4 mmol/l). A renal biopsy specimen revealed hyalinized glomeruli, tubular atrophy and interstitial inflammation.

Peritoneal dialysis and antihypertensive therapy were started and during the following months blood pressure returned to normal values, though no improvement of the renal function was seen. Renal transplantation was planned, and in November 1978 she received a kidney from a dead donor. The transplantation was successful, giving good kidney function.

In February 1979 she was referred to the outpatient department of dermatology for treatment of a plantar wart. On admission the elbows showed an increased carrying angle, with extension limited by 15 degrees and supination by 30 degrees (Fig. 1). All finger nails were dysplastic, the ulnar half of the thumb nails being most affected (Fig. 2). A V-shaped lunula was seen in all finger nails. All toe nails were abnormal, with ridging and splitting (Fig. 3). The patellae were hypoplastic. X-ray examination of the pelvis showed no signs of iliac horns.

The patient’s mother had been examined in another hospital some years previously and defects of the elbows and nails similar to those found in her daughter were registered. In addition she had had asymptomatic proteinuria for many years. The proband’s grandmother had died of a renal disease of unknown etiology, but information was not obtainable on nail or bone deformities. The proband’s father and single brother were said to be normal and were consequently not examined.

DISCUSSION

The nail–patella syndrome is a rare disorder and only few cases have been reported from the Scandinavian countries (5, 6, 7). The skeletal anomalies of the disease are not complicated by any significant disability. Renal involvement has been reported to occur in up to 42% of such cases (2). In most cases renal involvement begins with asymptomatic proteinuria, though nephrotic syndrome and progression to renal failure have also been observed (3, 4, 8, 10).

Recently, Bennett et al. (1) have described the pathologic symptoms of the kidneys in a large series of patients with nail–patella syndrome. Their light microscopical findings varied from normal to chronic proliferative glomerulonephritis showing correlation to the degree of renal dysfunction. However, by electron microscopy all 12 patients had abnormalities of the glomerular basement membrane. This was demonstrated even in 6 patients with completely normal light microscopy findings.

In our patient only light microscopy examination was done, revealing severe tubular atrophy and hyalinized glomeruli.

Terminal renal disease seems to be a rare complication in patients with nail–patella syndrome, and

**Fig. 1. Increased carrying angle of the elbows.**
only two cases of successful renal transplantation have been published (4, 8). Eisenberg et al. (4) did not biopsy the donor kidney. In the patient described by Uranga et al. (8) a biopsy of the transplanted kidney was performed one year after transplantation and most glomeruli were found normal by light microscopy. Electron microscopy was not done. In the case presented here, a biopsy of the donor kidney was not performed.

The underlying defect in the nail-patella syndrome has to be established. Provided that the defect in an inherited error of metabolism involving an enzymatic abnormality, a transplanted kidney might supply and replenish the enzyme, thus avoiding renal disease in the transplanted kidney. If, however, a humoral factor were responsible, the renal disease would recur in the transplant. Consequently it should be of interest to perform serial kidney biopsies in patients suffering from nail-patella syndrome and receiving renal transplantation for terminal renal disease. Since the skeletal deformities cause so few clinical problems, the impor-

Fig. 2. Dysplasia of finger nails (thumbs most affected) and V-shaped lunulae.

Fig. 3. Dysplasia of toe nails, with ridging and splitting.
tance of recognition of the syndrome lies with the associated nephropathy.

REFERENCES


Multiple Kerato-Acanthoma: A Case Report

G. Lange Wantzin, N. Agdal and E. Svejgaard

Department of Dermatology, University of Copenhagen, Rigshospital, Copenhagen, Denmark

Received April 8, 1980

Abstract: A case of multiple keratoacanthomas in a 36-year-old healthy seaman is presented. Four months prior to the onset of the disease the patient had been exposed to strong sunlight. Histopathological examination gave the diagnosis of keratoacanthoma.

Key words: Multiple keratoacanthoma

Keratoacanthoma was first described by Hutchinson in 1889 as the “crateriform ulcer of the face” (8). Later, Ferguson Smith (5) reported on a patient with “multiple self-healing squamous cell epithelioma”. However, it was not until 1950 that keratoacanthoma was established as a separate disease entity (10).

Keratoacanthoma is usually seen in the solitary form. This lesion is seen as a dome-shaped reddish tumour, not fixed to the underlying structures. The tumour enlarges up to 8 weeks and is characterized by a horny plug or a crust in its centre. Spontaneous regression usually occurs after a while and takes a few months (11). Keratoacanthomas may be eruptive (2, 11) and dissemination of the lesion has been reported in some cases (2).

CASE REPORT

A 36-year-old seaman was referred to this department in 1979 under the diagnosis recurrent folliculitis. Hitherto the patient had been in good health, and had no relevant family history of disease. Twenty years earlier he had had acne localized to the back. The present disease started 14 months before admission, with red itching papules on the lower legs, slowly spreading to the femora and gradually to the trunk and upper extremities. The single lesions remained unchanged for weeks and showed spontaneous healing, leaving a depressed scar. Previous topical corticosteroid treatment and peroral tetracycline had had no effect.

Four months prior to the onset of the disease the patient had been at sea in the tropics. Previous examinations in numerous countries including Holland and Canada had failed to establish a diagnosis.

Clinical examination revealed multiple tumours located to the extremities, including the soles, trunk, and ears. The tumours were firm reddish papules with central horny plugs or crusts. Some of the tumours were regressing. The size varied between 5 and 15 mm in diameter. Dispersed depressed scars were seen, probably left after tumour regression (Figs. 1 and 2). Tumours on the back, right arm, and left sole were examined microscopically.

Histopathology

The epidermis was hyper- and parakeratotic and slightly acanthotic. In the middle of the surface a keratin-filled crypt was seen. Strands of epidermis protruded into the dermis. The epidermis showed keratinization and proliferation and contained cells with “glassy” appearance. In the surrounding dermis a pronounced inflammatory infiltrate was seen, consisting of lymphocytes, granulocytes and plasma cells and, in addition, a few eosinophilic granulocytes. The vessels were normal, with perivascular cell infiltrates (Fig. 3).

Laboratory investigation

Blood counts and urine analysis were normal, as was X-ray of the lungs.