

Acquired Perforating Collagenosis in a Patient with Carcinoma of the Prostate

Sir,

Acquired perforating collagenosis is a rare skin disorder, mainly described in patients with severe renal failure and/or diabetes and rarely reported in patients with malignancies (1). We report on a patient who developed typical skin lesions 5 months after the diagnosis of a still untreated carcinoma of the prostate, tumour stadium pT2 N0 M0. Only mild renal insufficiency was diagnosed. No diabetes was found. Histology showed transepidermal elimination of degenerated collagen with necrotic destruction of the epidermis. Pruritus and consecutive scratching, microangiopathy, dermal microdeposits of crystals and proteolytic enzymes of leukocytes are believed to play a role in the pathogenesis of this perforating skin disorder. Therapeutic attempts described include phototherapy, topical keratolytics, steroids and retinoids.

CASE REPORT

A 64-year-old male Caucasian patient had been suffering from skin eruptions since April 1996 (Fig. 1). He had no history of previous



Fig. 1. Symmetrically distributed papules and nodules on both legs. The lesions show necrotic and keratotic material at their centres.

skin disease. The skin lesions started with strong pruritus, which subsequently decreased. In December 1995 a carcinoma of the prostate was diagnosed. Due to lack of compliance, the patient did not receive any treatment at that time. Additionally, he had been diagnosed as having a coronary heart disease with congestive heart failure and had undergone a coronary bypass graft 4 years earlier.

Physical examination revealed a patient in poor general condition, with evident signs of congestive heart failure. At the rectal examination it was discovered that a nodule of approximately 2 cm was palpable in the left proximal part of the prostate. Skin examination showed symmetrically disseminated red papules and nodules with necrotic and keratotic material at the centres of the older lesions. The lesions were distributed on the extremities and on the trunk, particularly on the shoulders and in the sacral region (Fig. 1). Prostate-specific antigen (PSA) (13.2 ng/ml) and β_2 -microglobuline (3, 3 mg/l) showed pathological values. Serum urea nitrogen was slightly elevated (34 mg/dl). The creatinine clearance was decreased to 64 ml/min. Further laboratory findings, including serum creatinine, serum glucose and urine sediment, were in the normal range. A prostate biopsy revealed a moderately differentiated cribriform carcinoma. In extensive tumour staging no lymph node, bone or organ metastases were detected. The prostate carcinoma was classified as pT2 N0 M0 G2. Skin biopsies showed transepidermal elimination of basophilic, degenerated collagen, with necrotic destruction of the epidermis. In the epidermis an infiltration of partly necrotic leukocytes was found. In the dermis a perivascular lymphohistiocytic infiltration was detected.

The skin lesions were first treated with 10% salicylic acid in petrolatum and then with topical steroids in a fatty base and moist compresses. After cardiac recompensation the patient received UV-radiation with combined UVA- and UVB-spectrum. There was a significant improvement of the skin condition. Treatment of the prostate carcinoma was started with complete androgen blockade with the testosterone antagonists Flutamide and Busereline. The androgen blockade has been continued up to now. Improvement of skin lesions began even before the initial dose for androgen blockade. Restitution was completed after 2 months of therapy with UV-radiation, topical keratolytics and topical steroids. During an 8-month follow-up the patient did not develop new skin lesions.

DISCUSSION

The case presented shows a new association of acquired perforating collagenosis with a solid tumour. Five months after diagnosis of a cribriform, up to that time untreated prostate carcinoma the patient had developed the typical skin lesions of a perforating dermatosis. Only a mild renal insufficiency was diagnosed. No diabetes mellitus was detected.

Up to now some thirty patients with acquired perforating collagenosis have been described. Most of them had diabetes mellitus and/or a severe renal failure. In a small number of patients no diabetes or renal insufficiency was found. Out of these patients 2 had Hodgkin's disease (2), one suffered from a mixed histiocytic-lymphocytic lymphoma (3), one was described with concomitant hypothyroidism and atopic eczema and one patient suffered from a systemic lupus erythematosus, chronic obstructive pulmonary disease and hepatopathy (4). Recently a report on a patient with an underlying liver carcinoma described the association of this disorder with another solid malignant tumour (5).

The exact pathogenesis of acquired perforating collagenosis is still unclear (1). The first attempts to explain the pathogenetic mechanism by a mild superficial trauma are still relevant; this is considered at least as a cofactor for the development of the skin lesions (1, 4). Obviously all described diseases like diabetes, renal failure, lymphoma, liver malignancy and atopic eczema may be associated with pruritus. In fact nearly all patients reported a mild to severe pruritus. In patients with diabetes microangiopathy was considered as a further causative factor.

As therapeutic modalities phototherapy, topical salicylic acid, topical and oral tretinoin application and topical steroids are described (1). In the 2 patients with Hodgkin's disease the skin lesions disappeared in one case after chemotherapy. In the other case chemotherapy had no effect (2). In our patient skin lesions improved after 3 weeks of therapy with topical salicylic acid, topical steroids and phototherapy with combined UVA- and UVB-spectrum. Because of the poor general condition the patient did not receive a radical prostatectomy but a complete androgen blockade. Subsequent radiation was planned.

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