

of an adenocarcinoma was not ruled out. The question arises whether the adenocarcinomas accompanying malignant acanthosis nigricans are in any way different from adenocarcinomas without malignant acanthosis nigricans. It was, therefore, with great interest that Hage & Hage's (3) findings of APUD (Amino Precursor-Uptake and Decarboxylation) cells in a gastric cancer of a patient with malignant acanthosis nigricans were received.

Hage & Hage (3) described a woman with widespread acanthosis nigricans (Fig. 1) and a gastric cancer of the diffuse and infiltrating type. Many tumor cells were identified by means of histochemistry and electronmicroscopy as neoplastic enterochromaffin-like cells. The authors suggested that cancers associated with malignant acanthosis nigricans might constitute a specific group of adenocarcinomas in which cells from parts of the tumor arise from the APUD-series of endocrine cells.

Therefore, the adenocarcinomas of 2 of my patients with malignant acanthosis nigricans were subjected to a search for cells of the APUD-series.¹

I. I. E., an 84-year-old Negro (4), had malignant acanthosis nigricans. When the dermatosis was discovered, a search for the tumor was undertaken. A carcinoma of the sigmoid colon (Figs. 2 and 3) was found and surgically removed before it caused any symptoms. The dermatosis cleared up immediately and he lived another 8 years. He died at the age of 92 years from congestive heart failure.

2) A. B., a 60-year-old black woman, had widespread acanthosis nigricans. At Presbyterian Hospital in New York an annular gastric cancer (Fig. 4) was visualized and gastrectomy was performed. There were metastases to many lymph nodes. After the operation the dermatosis remained unchanged. The patient died 9 months following gastrectomy.

New sections of these two tumors were stained with the methods of Fontana and the methods of Grimelius. They highlight, respectively, argentaffin granules and argyrophilic granules, that is, those intracellular, neurosecretory, granules which are associated with cells of the APUD system. The results of these stains were entirely negative in the adenocarcinomas, while they were positive in the overlying mucosa, which served as a built-in control, since APUD cells are normally found in these tissues.

It therefore seems that Hage & Hage's findings of APUD cells in adenocarcinomas accompanying

malignant acanthosis nigricans are not characteristic of tumors associated with malignant acanthosis nigricans, but rather a chance occurrence. Moreover, adenocarcinomas of the gastro-intestinal tract without accompanying malignant acanthosis nigricans may contain smaller or greater numbers of cells with argentaffin and/or argyrophilic granules (5) and some small cell "undifferentiated" colonic cancers derive from APUD elements (6).

It would seem advisable to examine additional cancers accompanying malignant acanthosis nigricans for APUD cells to evaluate further the findings of Hage & Hage.

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Solitary Lichen Planus Simulating Malignant Lesions

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Abstract. Four patients with similar cutaneous lesions clinically simulating Bowen's disease or basal cell carcinoma revealed histopathological changes of lichen

¹ I am very grateful to Dr Raffaele Lattes, who supervised the examinations.

planus. Examination by direct immunofluorescence was performed in two of the patients, showing ovoid bodies containing IgM, IgA and fibrinogen in one patient and IgM and fibrinogen in the other patient.

Key words: Lichen planus; Lichenoid keratosis; Bowen's disease; Basal cell carcinoma

In 1976 Scott & Johnson (5) reported a large series of patients with solitary cutaneous lesions appearing clinically as papules or plaques showing histopathological changes of lichen planus. The same entity seems to have been reported as "solitary lichen planus" (2), "solitary lichen planus-like keratosis" (6), and "lichenoid actinic keratosis" (1). The macroscopic diagnosis in these cases was that of keratosis, basal cell carcinoma, Bowen's disease, or cellular naevus but in no case was it lichen planus. Very few photographs have been published of this lesion (1).

Four patients will be described with clinically almost identical solitary lesions simulating malignant lesions but histopathologically demonstrating a benign picture of lichen planus.

CASE REPORTS

Case 1

A 65-year-old woman presented with a mildly itching solitary lesion located on the ventral part of her left lower leg for half a year. The lesion was erythematous, scaling, non-infiltrated and sharply defined with a diameter of 1 cm (Fig. 1). The preliminary clinical diagnosis was Bowen's disease. The histopathological examination showed slight hyperkeratosis, focal acanthosis and degeneration of the basal cell layer. In the upper dermis there was an infiltrate of histiocytes and lymphocytes invading the epidermis. The histological diagnosis was lichen planus.

This patient was seen again after 5 months. Spontaneous regression of the lesion had occurred, leaving only hyperpigmentation. Examination by direct immunofluorescence revealed cytooid bodies containing IgM, IgA and fibrinogen immediately under the basement membrane zone. The patient had no other skin, mucosal or nail lesions.

Case 2

A 66-year-old woman presented with a mildly itching solitary lesion on her upper chest, having been present for 2 months. The lesion was erythematous with slight scaling, and was 1 cm in diameter (Fig. 2). The preliminary clinical diagnosis was Bowen's disease or basal cell carcinoma. The lesion was excised. The histopathological examination, however, demonstrated hyperkeratosis, slight parakeratosis, acanthosis, degeneration of the basal cell layer plus an infiltrate of histiocytes and lymphocytes immediately below the epidermis. The histological diagnosis was lichen planus.

Case 3

A 54-year-old woman presented with a mildly itching solitary lesion on the ventral part of her left lower leg, having been visible for one year. The lesion was erythematous and scaling, 13×12 mm in size (Fig. 3). The preliminary clinical diagnosis was Bowen's disease. The histopathological examination showed thickening of the granular layer, degeneration of the basal cell layer and, in the upper dermis, proliferation of the capillaries and an infiltrate of predominantly lymphocytes. The histological diagnosis was lichen planus.

This patient was seen again 2 months later after treatment with a topical steroid. Only hyperpigmentation remained. Examination by direct immunofluorescence was now performed, showing cytooid bodies containing IgM and fibrinogen immediately under the basement membrane zone. No other changes of skin, nails or mucosa were demonstrated, except for a lacy pattern of milky white papules on the inside of her right cheek, typical of lichen planus. Five years earlier she had had multiple skin lesions on her arms and legs clinically and histologically diagnosed as lichen planus, which disappeared after 4 weeks.

Case 4

An 80-year-old man presented with an intermittent ulcerating solitary lesion localized to the left of the umbilicus. It had been observed over a period of about 4 years with slow progression and, at examination was, 2 cm in diameter, erythematous and scaling (Fig. 4). There were no other changes in skin, nails or mucosa. The preliminary clinical diagnosis was Bowen's disease or basal cell carcinoma. The lesion was treated with curettage and electrocoagulation. The histopathological examination showed hyperkeratosis, slight parakeratosis, degeneration of the basal cell layer and, in the upper dermis, an infiltrate of lymphoid cells (Fig. 5). The histological diagnosis was lichen planus.

COMMENTS

The lesions reported macroscopically simulated Bowen's disease in two cases and Bowen's disease or superficial basal cell carcinoma in the other two cases. In no case was there any clinical similarity to the normal picture of lichen planus, not even when the histopathological findings became known.

The lesions may nevertheless constitute a special type of genuine lichen planus. The findings of lichen planus lesions in the mucosa of the mouth in one of the patients (case 3) and the earlier history of a classical lichen planus in the same patient support such a view. The immunological findings in the immunofluorescence studies also agree with these considerations (4).

It has been proposed that the lesion might be a variant of actinic keratosis (1, 3). However, this

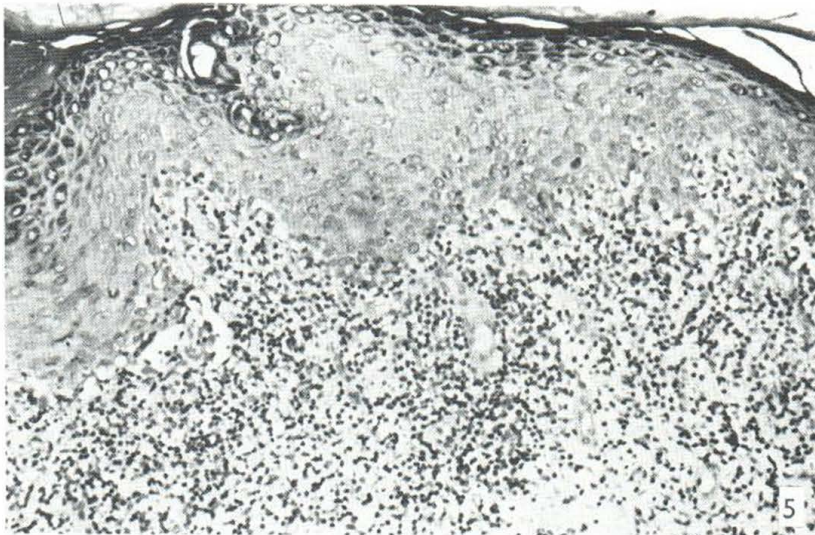
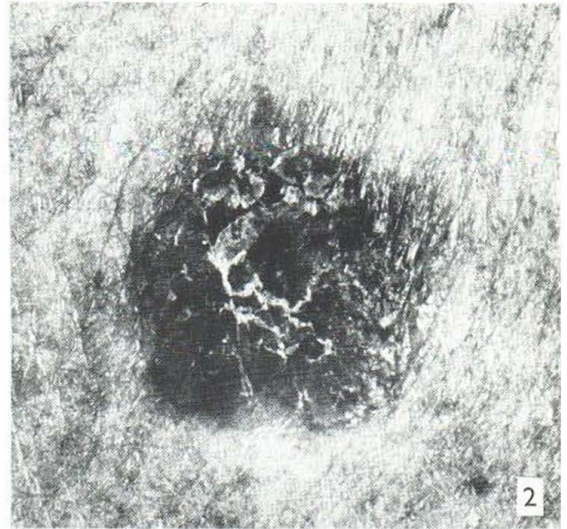
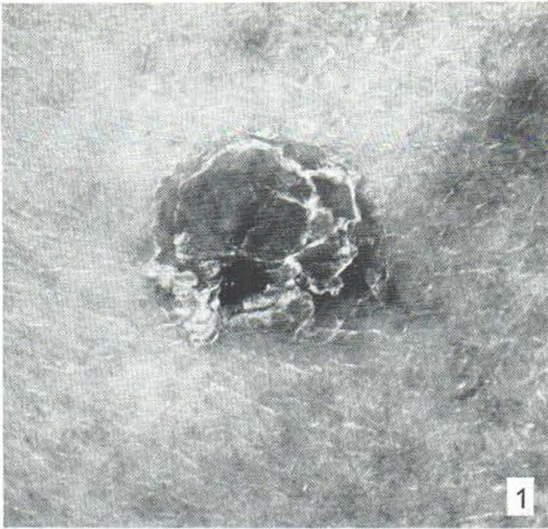


Fig. 1-4. Solitary lesion on the:

1. Lower leg.
2. Upper chest.
3. Lower leg.
4. Abdomen.

Fig. 5. Microscopic picture of solitary lesion on the abdomen.

was not supported by the microscopic findings in the present cases and, furthermore, in 2 of the patients the localization was in non-sun-exposed areas.

These 4 patients were seen over a 4-month-period, which may indicate that this disease simulating malignant lesions is not really so uncommon.

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Synchronous Balding of Scalp and Hair-bearing Grafts of Scalp Transplanted to the Skin of the Arm in Male Pattern Baldness

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Abstract. The author transplanted composite skin grafts from balding, non-balding, and bald areas of the scalp, to the skin of the arm. The galea aponeurotica was trimmed away from the grafts. The patient was a 29-year-old male with progressive male pattern baldness (MPB).

The transplants from the balding area became bald at the same rate as the balding donor site in the receding frontal hairline, whereas the transplants taken from the non-balding area in the occiput continued to grow the same amount and quality of terminal hairs. Bald grafts taken in front of the receding hairline remained bald. This shows that the cause of MPB lies in the follicle itself or in

its very close surrounding and does not depend on the galea aponeurotica, the increased tension of the scalp or of its muscles, the diminished vascular supply to the scalp or any other regional factor localized to the head area. It also shows that the "balding clock" keeps time even when the follicle is transplanted to another region of the body.

Key words: Male pattern baldness; Galea aponeurotica; Alopecia; Hair transplantation

During the last few decades, several hypotheses concerning the etiology of male pattern baldness (MPB) have been presented. In 1933, Wadel (10) reported findings of decreased motility of the scalp. He was convinced that this decrease was due to the fact that in MPB patients the scalp is both frontally and sagittally too short, and thus it has to be stretched like a too-small cap to cover the relatively too-big skull. For hair nutrition and rooting this persisting tension creates unbearable conditions, leading to gradual loss of hair. In 1935 (11) he wrote that MPB is the end result of the tension atrophy of the scalp covering the galea aponeurotica. This atrophy is caused by a disproportion between the skull bone and the galea aponeurotica, due to an isolated growth of the skull bone to which the tendon-like structure of the galea is not able to adapt. He reported excellent results in the treatment of MPB with "loosening" massage to the scalp.

In 1941, Kessler (2) started experimental work with frontal galeotomies in order to reduce the supposed increased tension of the galea aponeurotica. In 1961 he reported (3) a success rate of 87% with this treatment of MPB. At that time this operation was popular in Europe. In 1963, Pontén (8) reported that after frontal galeotomy he could not find any objective improvement in his 56 patients and he still holds this view concerning this operation (personal communication, 1976).

The present author has seen several patients who have undergone frontal galeotomy and later developed an advanced degree of MPB. The popularity of this operation has waned.

A still-popular idea concerning the etiology of MPB is the decreased vascular supply to the scalp (5, 10). Dorsey (1) considered this to be due to the trauma produced by tightly fitting hats to the temporal artery and vein.

Szasz & Robertson (9) suggested that the increased tension of the scalp muscles is the cause of MPB.