Acta Derm Venereol (Stockh) 1994; 74: 449–450

Pseudo-fibrokeratoma of the Nail Apparatus with Melanocytic Pigmentation: A Clue for Diagnosing Bowen's Disease

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Bowen's disease of the nail apparatus is a protean condition where pseudo-fibrokeratoma associated with melanocytic pigmentation was the clue leading to the clinical diagnosis. Migration of melanocytes into the superficial layers of the matrix epithelium may be associated with the extension of the epidermal tumoral process, without necessitating a real melanocytic hyperplasia.

(Accepted April 11, 1994.)

Acta Derm Venereol (Stockh) 1994; 74: 449–450.

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We report an unusual clinical feature of ungual Bowen's disease masquerading as acquired fibrokeratoma associated with melanocytic colonization.

CASE REPORT

An 80-year-old Caucasian male sought advice for a lesion beneath his left finger nail which had been present for more than 2 years. Examination revealed the nail plate to be slightly lifted radially by a tumour identifiable through the nail as a longitudinal melanonychia (Fig. 1). The distal portion of this dark keratotic lesion was evident under the ungual free edge. The patient had no occupational history of X-ray exposure, psoriasis or recollection of single or repeated trauma.

After latero-longitudinal biopsy excision, the most prominent feature seen histologically on low power was a marked papillomatosis of the nail bed with distally finger-shaped architecture ending at the distal groove and covered by thick keratin pushing up the nail plate. The nail bed architecture was in complete disorder. The tumoral process extended to the nail matrix and was sharply demarcated at the border of normal proximal nail matrix (Fig. 2). The epithelial cells had hyperchromatic nuclei varying in size and shape. There were multinucleated cells, dyskeratotic cells and frequent abnormal mitotic figures (Fig. 2).

On Fontana-Masson stain, melanocytes were randomly dispersed as a network among neoplastic cells with insinuating dendritic processes outlined by fine melanin granules (Fig. 3). No perikaryon of melanocytes was detected in the lowest layer of the nail matrix. The melanocytes were decorated by S 100 protein and HMB 45. The melanin granules were abundant in the more superficial epithelial cells of the nail matrix, as opposed to the lower layers, where the granules were dispersed along the dendrites of melanocytes.

Some granules were incorporated in the lower third of the nail plate.

The melanocyte count of the matrix and the nail bed, as well as the calculation of the mean interval separating two melanocytes, are shown in Table I. This calculation was based on five vertical sections each 5 μ thick, using a micrometer. No melanocytes were detected in normal proximal matrix on Fontana-Masson, S 100 protein, HMB 45.

Fig. 1. Clinical presentation of the subungual tumour.

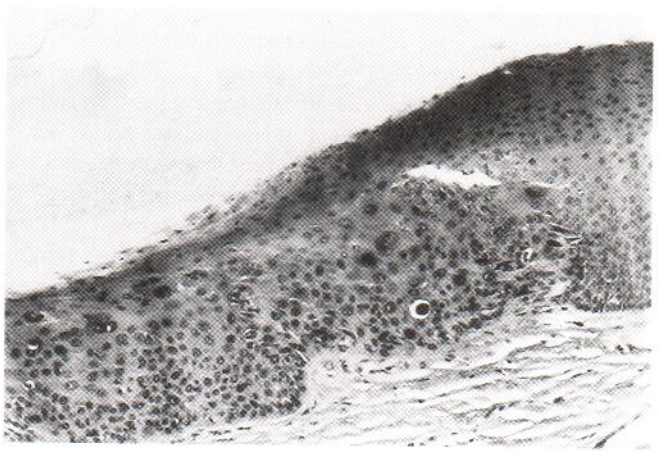

Fig. 2. Bowen's tumoral process in the left portion of the matrix with sharp delineation, contrasting with the normal matrix on the right portion (HES stain × 10).

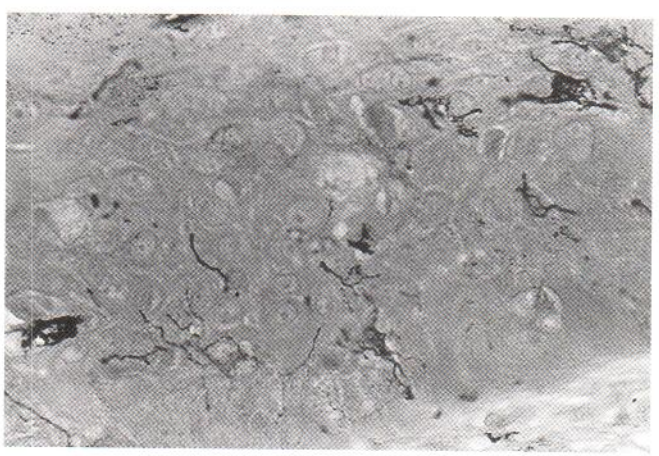

Fig. 3. Dendritic melanocytes in the tumoral portion of the nail matrix (Fontana-Masson stain × 40).

  ISSN 0001-5555

Table I. *Incidence and interval of melanocytes compared in previous and present studies*
PFK: pseudo-fibrokeratoma; max: maximum; min: minimum.

	Normal matrix (Higashi's studies)	Matrix of PFK	Nail bed of PFK
Interval of melanocytes on vertical sections	77,3 to 170,5 μ	90,7 μ (min. 25, max. 150)	112,5 μ (min. 725, max. 12550)
Melanocyte incidence			
Cell layers of epithelium	266 ± 26 mm²	40 ± 5 mm²	
Four lower layers of epithelium	334 ± 98 mm²	126 ± 20 mm²	16 ± 3 mm²

DISCUSSION

The combination of apparent longitudinal melanonychia visible through the nail and acquired fibrokeratoma led us to suspect Bowen's disease, in particular as Haneke (1) has described epidermoid carcinoma of the nail simulating acquired ungual fibrokeratoma. Colonization of non-melanocytic tumours by dendritic melanocytic cells is a well recognized phenomenon in melano-acanthoma (2, 3) and in breast carcinoma invading the epidermis (4). A few reports have underlined this phenomenon in malignant eccrine poroma (5), sebaceous adenoma (5), squamous cell carcinoma in situ of the oral mucosa (3) and ulcerated mucoepidermoid carcinoma arising on the lip (6). To our knowledge, such a colonization has been mentioned in only one case of pigmented Bowen's disease (7, 8). The present data offer more evidence of the ability of non-neoplastic dendritic melanocytes to pigment different types of tumours. We know that melanocytes have occasionally been found in the nail bed epithelium of blacks with hyperpigmented bands due to an increased activity of melanocytes without hyperplasia (9) but not in normal nail bed epithelium of whites or orientals (10). In our case, we have found very few stained melanocytes in the tumoral nail bed.

Melanocytes may undergo hyperplasia for this to occur, but, comparing melanocytic distribution and its number obtained on a normal epithelium with those noted in our case on neoplastic epithelium may lead to an alternative hypothesis.

The distribution of melanocytes in nail matrix differs from that in normal epidermis. The cells are found in the two to four first layers of the matrix but not in the lowest layer of nail matrix cells (11, 12). In our case the lowest layer of matrix cells was free of melanocytes, and the mean value of the intervals of melanocytes on vertical sections was similar to that found by Higashi (11) but with a total melanocyte count of 266/mm², which is less than that reported by Higashi & Saito (13) (Table I).

Even if Higashi's count was based on horizontal sections, there is, if we consider Cochran's work (14), a good correlation between the counts of melanocytes on horizontal and vertical sections. In addition, as in our case, Higashi's melanocyte counts do not take into account the proximal portion of the ventral matrix.

It is well established that no significant difference in the density of distribution of skin melanocytes exists between black, oriental and Caucasian individuals. Consequently, migration of melanocytes into the superficial layers of the matrix epithelium may be associated with the extension of the epidermal tumoral process, without necessitating a real melanocytic hyperplasia.

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