



Dermoscopy as a Supportive Instrument in the Early Recognition of Erosive Adenomatosis of the Nipple and Mammary Paget's Disease

Enzo Errichetti, Claudio Avellini¹, Enrico Pegolo¹, Vincenzo De Francesco

Department of Experimental and Clinical Medicine, Institute of Dermatology, University of Udine, ¹Department of Medical and Biological Sciences, Institute of Anatomic Pathology, University of Udine, Udine, Italy

Dear Editor:

Erosive adenomatosis of the nipple (EAN) and mammary Paget's disease (MPD) are respectively a benign and a malignant neoplasm affecting the nipple/areola^{1,2}. As the clinical characteristics of these conditions are usually non-specific and variable, they are often mistaken for inflammatory skin diseases, such as psoriasis and eczema^{1,2}. Recently, it has been shown that, in long-standing cases, dermoscopy may be used as an auxiliary tool in assisting their recognition^{1,2}. However, there is no data on dermoscopy of such disorders in their initial stages, when the clinical identification is even more challenging. We here describe the dermoscopic features of two early instances of EAN and MPD.

Case 1. A 41-year-old woman presented with a 1-month history of a subtle asymptomatic erosion associated with moderate hyperkeratosis on the right nipple (Fig. 1A). On noncontact polarized light dermoscopic examination, such lesion showed whitish/yellowish hyperkeratosis and sparse dotted vessels on a reddish-whitish background (Fig. 1C). Histology displayed typical features of EAN (Fig. 1E).

Case 2. An 88-year-old woman was referred to our clinic with a 1-month history of a small asymptomatic serohe-matic crust on the right nipple (Fig. 1B). Noncontact polarized light dermoscopy displayed a brownish-reddish crust associated with linear irregular vessels on a pinkish-whitish background (Fig. 1D). Histology and immunohisto-

chemical analysis yielded a diagnosis of MPD (Fig. 1F).

The use of dermoscopy for assisting the diagnosis of skin conditions other than melanocytic lesions/common non-melanocytic tumours has remarkably increased over the last years³. Regarding MPD, most of the attention has been focused on the pigmented variant, with diffuse irregular pigmentation, regression-like structures (especially gray-blue structures) and irregular vessels being described as the commonest findings by several reports^{4,5}. According to a recent instance, some of the above-mentioned features may be found even in the classic (clinically nonpigmented) form of MPD, including light brown diffuse pigmentation, irregular black dots, irregularly distributed blue-gray dots (peppering), and irregular linear vessels; moreover, several chrysalis-like structures may additionally be detected¹. On the other hand, based on a recent case report, dermoscopic examination of EAN may show cherry-red dots over a reddish background and non-specific peripheral orange collar-like veils, interpreted by the authors as luminal openings and remaining epidermis, respectively².

In the present two instances, we observed some of the findings reported in the two aforementioned long-standing cases of MPD and EAN, namely irregular linear vessels and sparse dotted red structures (which we interpreted as dotted vessels as they disappeared after pressing the dermoscope), respectively. Such a difference in dermoscopic vascular patterns might be explained by the well-known

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Corresponding author: Enzo Errichetti, Institute of Dermatology, "Santa Maria della Misericordia" University Hospital, Piazzale Santa Maria della Misericordia, 15. 33100-Udine, Italy. Tel: 39-0432559820, Fax: 39-0432559892, E-mail: enzoerri@yahoo.it

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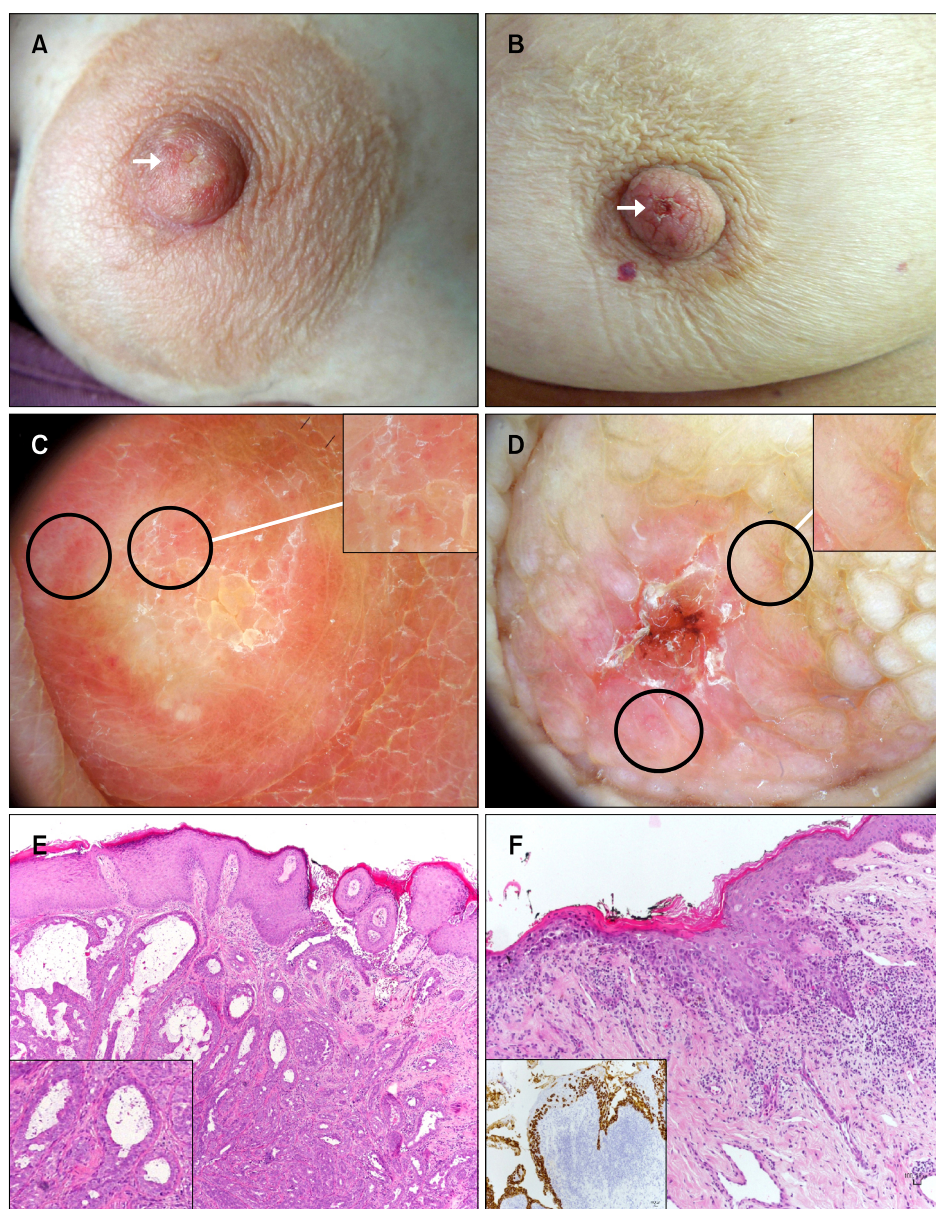


Fig. 1. Clinical examination of the right nipple shows a slight erosion (arrow) associated with moderate hyperkeratosis in the first patient (A) and a small serohematic crust (arrow) in the second patient (B). Dermoscopic examination (carried out with DermLite DL3×10; 3Gen, San Juan Capistrano, CA, USA) of the right nipple in the first case displays whitish/yellowish hyperkeratosis and some dotted vessels (circles) on a reddish-whitish background (C), while it shows a brownish-reddish crust associated with linear irregular vessels (circles) on a pinkish-whitish background in the second case (D). Histologic examination in the first patient reveals typical features of erosive adenomatosis of the nipple, namely ductal structures in the dermis focally connected to the overlying epidermis (H&E, ×40); these ducts result mostly lined with a basal layer made up of myoepithelial cells and a luminal layer of epithelial cells exhibiting apocrine differentiation (box) (H&E, ×200) (E). In the second patient, histology shows large atypical cells with hyperchromatic eccentric nuclei and abundant cytoplasm throughout the epidermis, hyperkeratosis, parakeratosis, and acanthosis (H&E, ×200); immunohistochemical analysis demonstrates that the tumour cells are positive for cytokeratin 7 (box) (F). All these findings are consistent with the diagnosis of Paget's disease.

concept that benign lesions commonly present a regular vascularity and malignant lesions usually display more irregular vessels^{1,2}. In our opinion, the detection of the above-mentioned findings might help the clinician raise

the suspicion of such conditions (with consequent prompt biopsies) over the main differential diagnoses, i.e., eczema and psoriasis, as they usually show different features, namely yellowish serocrusts/patchily distributed dotted

vessels and whitish scales/diffusely distributed dotted vessels, respectively³. Obviously, further studies on larger groups of patients are needed to confirm our observations.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Attenuated Nuclear Factor Kappa B Activity by E7 Protein of Human Papillomavirus Type 2 in Human Epidermal Keratinocytes

Yun Hee Ryu, Ju Hee Han, Ji Hyun Lee, Soon Young Choi¹, Young Min Park

Department of Dermatology, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, ¹Department of Biological Sciences and Biotechnology, Hannam University, Daejeon, Korea

Dear Editor:

Human papillomavirus (HPV) is a double stranded DNA virus that induces hyper-proliferative lesions of cutaneous and mucosal epithelium. HPV is composed of six early genes (E1, E2, E4, E5, E6, and E7) and two late genes (L1 and L2). Early genes are related to viral replication and transcription whereas late genes encode viral structural proteins^{1,2}. The E6 and E7 genes are major oncoproteins which are associated with the tumor suppressor proteins, p53 and retinoblastoma protein. In addition, the E7 gene has been known to play a role in evading host immune re-

sponses³.

Toll-like receptors (TLRs) are the first line of defense in host protection against invasion of microbial pathogens. TLR signaling pathways induce the transcription factor nuclear factor kappa B (NF- κ B)⁴. Among 10 human TLRs, TLR3 and TLR9 are known to be upregulated in wart lesions⁵.

Common warts caused by type 2 HPV (HPV2) are clinically characterized by little inflammation. Thus, we used to adopt a kind of immunotherapeutic modalities including diphenylcyclopropenone and imiquimod, which can

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Corresponding author: Young Min Park, Department of Dermatology, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, 222 Banpo-daero, Seocho-gu, Seoul 06591, Korea. Tel: 82-2-2258-6223, Fax: 82-2-594-3255, E-mail: ymmpark6301@hotmail.com

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