

SHORT COMMUNICATION

Dermoscopic Features of Plasma Cell Cheilitis and Actinic Cheilitis

Takamasa Ito¹, Ken Natsuga^{1*}, Shintaro Tanimura², Satoru Aoyagi¹ and Hiroshi Shimizu¹

Departments of Dermatology, ¹Hokkaido University Graduate School of Medicine, N15W7, Kita-ku, Sapporo 060-8638, and ²Chitose City Hospital, Chitose, Japan. E-mail: natsuga@med.hokudai.ac.jp

Accepted Nov 7, 2013; Epub ahead of print Jan 28, 2014

Plasma cell cheilitis (PCC) is a rare, idiopathic, and benign inflammatory mucosal condition characterised by dense polyclonal plasma cell infiltrates within the mucosa (1). It usually presents as a circumscribed, flat to slightly raised, eroded plaque or patch, usually on the lower lip of elderly individuals (1). The first reported case of plasma cell mucositis involving a mucocutaneous junction was plasma cell balanitis described by Zoon in 1952 (1). Subsequent reports disclosed similar conditions in other mucosal areas, including plasma cell vulvitis, gingivitis, and cheilitis (2). The cause is unknown, but some cases respond favourably to the application of topical steroids and oral griseofulvin (2).

Actinic cheilitis (AC) is a chronic inflammatory process caused by chronic exposure to sun light or artificial ultraviolet radiation and clinically presents as loss of the usually sharp border of the lip, atrophy of the vermilion border and darkening of the lip at the border between the lip and the skin of the face, as well as ulcers of the lip. AC affects the lower lip in 95% of the cases (3), as it is more directly exposed to sunlight. AC is also known as actinic keratosis (AK) of the lip and is considered a potentially malignant disorder likely to develop into invasive squamous cell carcinoma (SCC) as in the case of AK (4). The dermoscopic features of AK, such as strawberry pattern are well known (5). However, the dermoscopic features of AC have never been reported. This is probably because dermoscopic examination is not routinely performed before excision of the lip lesion.

Here we describe the typical dermoscopic features of PCC by comparison with those of the other type of cheilitis AC.

CASE REPORTS

Plasma cell cheilitis

Case 1. A 78-year-old man had noticed an erosive lesion on his lower lip for 4 years (Fig. S1a¹). Clinical examination showed well demarcated erythematous erosive plaques on his lower lip that histology subsequently confirmed as PCC (Fig. S1c,d¹).

Case 2. A 74-year-old woman had noticed a demarcated shallow ulcer with slight pain in her lower lip one year

before she was referred to our hospital. (Fig. S1b¹). The diagnosis of PCC was confirmed histologically, immunologically and serologically (data not shown).

Actinic cheilitis

Case 1. A 79-year-old man had complained of shallow ulcer with infiltrated border on his lower lip for 2 years (Fig. S2a¹). Histological specimens confirmed the diagnosis of AC (Fig. S2d¹).

Case 2. A 74-year-old man came to our hospital with a shallow ulcer with infiltrated border on his lower lip which he had noticed 6 months before referral (Fig. S2b¹). The diagnosis of AC was histologically confirmed (data not shown).

Case 3. The 74-year old man had noticed a shallow ulcer with infiltrated border on his lower lip for one year (Fig. S2c¹). The histopathology confirmed AC (data not shown).

RESULTS AND DISCUSSION

Disorders of the lips of the oral cavity are commonly designated as cheilitis, representing an inflammatory process that affects the vermilion of the lips. Many types of cheilitis appear clinically similar to PCC and AC, such as contact cheilitis and granulomatous cheilitis. AC deserves special attention because of its malignant potential to develop into invasive SCC of the lip (6). Once SCC develops on the lip, the risk of invasion and metastasis to the cervical lymph nodes is higher than that for SCC of the skin (7). Therefore, it is important to differentiate PCC from AC.

In our series, we observed dermoscopic typical features of PCC in comparison to those of AC. In both conditions, radially arranged vascular enlargement and proliferation around the ulcer were seen. Such vascular pattern was observed to a greater or lesser extent in the dermoscopic features of PCC; however, the border of PCC was clear and regular. In PCC, the whitish area had dense lymphocytic infiltration and degenerative epithelial changes (8) as seen in our 2 cases (Fig. 1). In contrast, the border of AC was irregular and ill defined, because of white-coloured projections and island-like structures with interspersed areas of red in colour. The whitish areas represent the hyperkeratotic region (9–11) which we confirmed histologically as hyperkeratosis

¹<http://www.medicaljournals.se/acta/content/?doi=10.2340/00015555-1795>

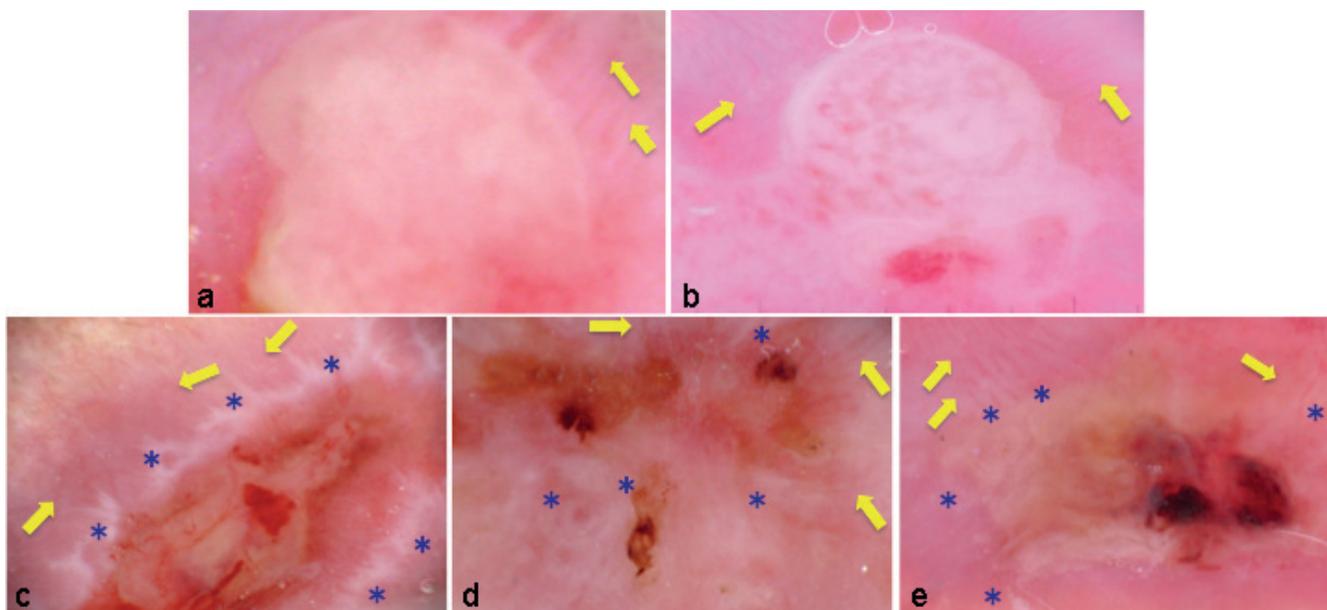


Fig. 1. Dermoscopy of PCC1 (a) and PCC2 (b) showing a regular and well-demarcated border with vascular telangiectasia (arrows). Dermoscopy of all 3 AC cases showing irregular and ill-defined borders with radially arranged vascular enlargement and proliferation around the ulcer (arrows) (c–e). Whitish projections and island-like structures representing hyperkeratosis were seen (asterisks).

in our AC cases. Hence these histological features are regarded as being responsible for the difference between PCC and AC. Whitish projections and island-like structures can be explained by the current concept of cancer stem cells. Stem cells in steady state are distributed to histological regular pattern in each organ (12). However, cancer stem cells are thought to be located randomly, suggesting aberrant pattern of differentiation. As whitish projections and island-like structures are supposed to be hyperkeratosis, cancer stem cells in AC might exist in a random manner to exhibit an asymmetrical and irregularly distributed cornified layer (12).

In conclusion, dermoscopy is a useful tool for evaluating PCC and AC. Border regularity with vascular enlargement and proliferation are diagnostics in PCC. On the other hand, dermoscopic characteristics of AC are ill-demarcated borders and vascular telangiectasia, white-coloured projections and island-like structures around the ulcerous areas. To our knowledge, this is the first dermoscopic study of PCC and AC.

The authors declare no conflict of interest.

REFERENCES

1. Zoon JJ. Balanoposthite chronique circonscrite benigne a plasmocytes (contra erythroplasia de Queyrat). *Dermatologica* 1952; 105: 1–7.

2. Jin SP, Cho KH, Huh CH. Plasma cell cheilitis, successfully treated with topical 0.03% tacrolimus ointment. *J Dermatol Treat* 2010; 21: 130–132.
3. Ron E. Ionizing radiation and cancer risk: evidence from epidemiology. *Radiat Res* 1998; 150: s30–41.
4. Picascia DD, Robinson JK. Actinic cheilitis: a review of the etiology, differential diagnosis, and treatment. *J Am Acad Dermatol* 1987; 17: 255–264.
5. Zalaudek I, Giacomel J, Schmid K, Bondino S, Rosendahl C, Cavicchini S, et al. Dermoscopy of facial actinic keratosis, intraepidermal carcinoma, and invasive squamous cell carcinoma. *J Am Acad Dermatol* 2012; 66: 589–597.
6. Vieira RA, Minicucci EM, Marques ME, Marques SA. Actinic cheilitis and squamous cell carcinoma of the lip: clinical, histopathological and immunogenetic aspects. *An Bras Dermatol* 2012; 87: 105–114.
7. Robinson JK. Actinic cheilitis: A prospective study comparing four treatment methods. *Arch Otolaryngol Head Neck Surg* 1989; 115: 848.
8. Baughman RD, Berger P, Pringle WM. Plasma cell cheilitis. *Arch Dermatol* 1974; 110: 725–726.
9. Markopoulos A, Albanidou-Farmaki E, Kayavis I. Actinic cheilitis: clinical and pathologic characteristics in 65 cases. *Oral Dis* 2004; 10: 212–216.
10. Cockerell CJ. Pathology and pathobiology of the actinic (solar) keratosis. *Br J Dermatol* 2003; 149: 34–36.
11. Ackerman AB, Mones JM. Solar (actinic) keratosis is squamous cell carcinoma. *Br J Dermatol* 2006; 155: 9–22.
12. Owens DM, Watt FM. Contribution of stem cells and differentiated cells to epidermal tumours. *Nat Rev Cancer* 2003; 3: 444–451.