

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Squamous Cell Carcinoma at the Site of Cutaneous Lymphoid Hyperplasia

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Dear Editor:

An 82-year-old Japanese female noticed an erosive plaque on her lower lip in 2012. Although the plaque healed by itself, it

recurred after a month. She was referred to our hospital in 2014. She did not smoke or drink alcohol. Her past history was not in particular, and she had no experience of radiation therapy, burns, herpes zoster, and insect bite on her lip. She presented with partially erosive plaque with surrounding bulges measuring about 1.0 cm×2.0 cm on her lower lip (Fig. 1A). The serum level of zinc was within the normal limit (71.9 µg/dl). Histopathological examination of a biopsy specimen from the erosive plaque showed the elongation of rete ridges and parakeratosis, pseudocarcinomatous epithelial hyperplasia, superficial and deep infiltration of plasma cells, and small lymphocytes forming lymphoid follicles (Fig. 1B, C). Immunohistochemical staining showed CD20 was positive in folli-

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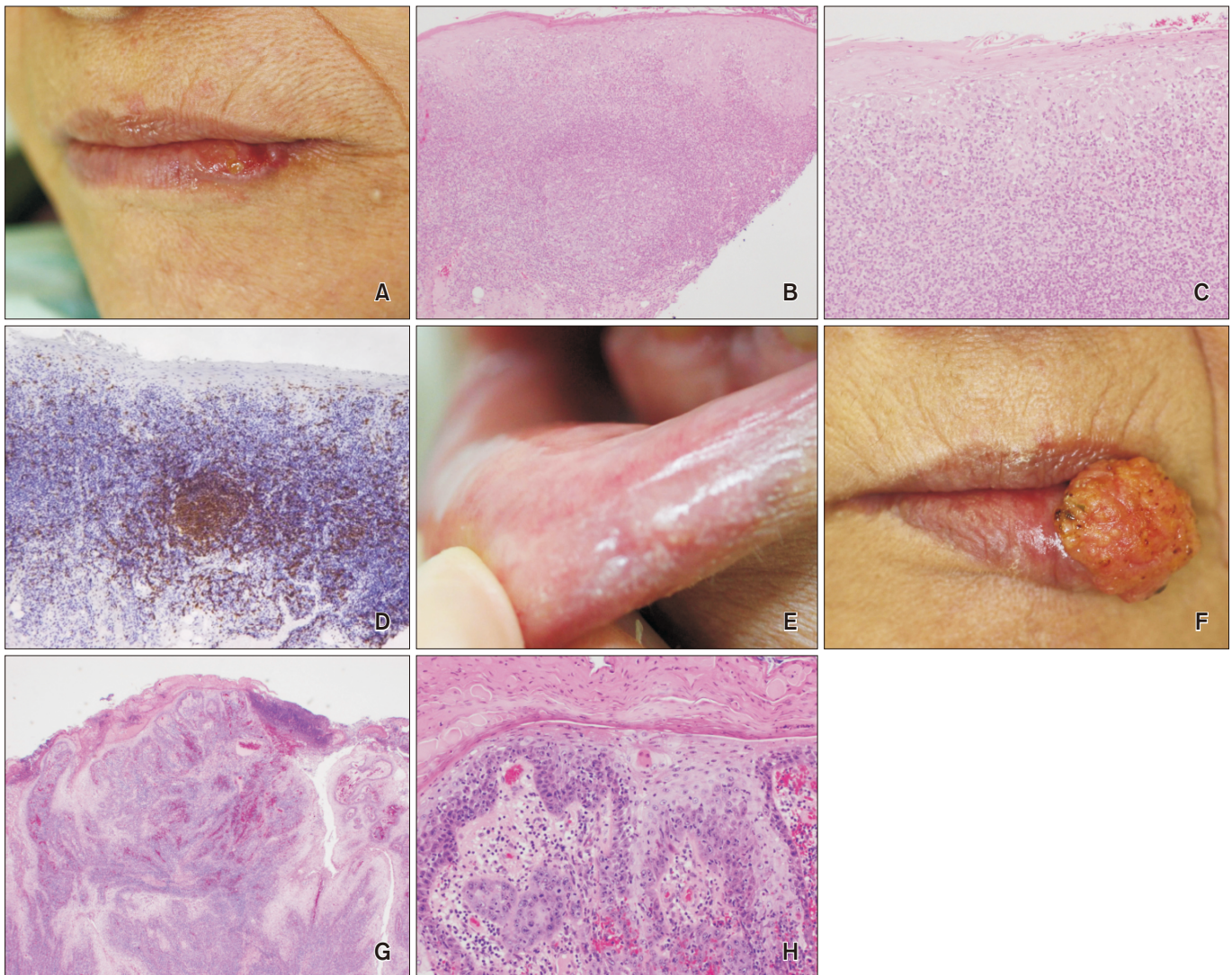


Fig. 1. (A) Clinical presentation at first visit (we received the patient's consent form about publishing all photographic materials). (B, C) Histopathological examination of a biopsy specimen from the erosive plaque showed the elongation of rete ridges and parakeratosis, pseudocarcinomatous epithelial hyperplasia, superficial and deep infiltration of plasma cells, and small lymphocytes forming lymphoid follicles (H&E; original magnification: B, $\times 40$; C, $\times 200$). (D) Immunohistochemical staining showed that CD20 was positive in follicular cells (original magnification, $\times 40$). (E) After 2 months, the erosions epithelialized. (F) A tumor measuring 3.0 cm \times 1.5 cm after 12 months at the same site. (G, H) Histopathological examination of the resected specimen showed proliferation of atypical keratinocytes invaded through the dermis with keratinization (H&E; original magnification: G, $\times 12.5$; H, $\times 200$).

cles (Fig. 1D), CD3, CD4, and CD8 were positive beside them, and CD30 was negative. *In situ* hybridization of κ and λ light chains did not show light chain restriction, which indicated polyclonal plasma cell infiltration. Clonal rearrangement for blood of T-cell receptor (TCR)-C β 1, TCR-J γ chain and immunoglobulin heavy chain genes using Southern blot analysis was not detected. We diagnosed her with cutaneous lymphoid hyperplasia (CLH) and decided watchful waiting¹. After 2 months, the erosions spontaneously epithelialized (Fig. 1E).

However, a plaque developed after 6 months at the same site, which develop to a tumor measuring 3.0 cm \times 1.5 cm after 12 months in 2015 (Fig. 1F). Histological examination of a second lip biopsy showed comparatively atypical keratinocytes that proliferate irregularly in the deep direction with keratinization. There was no finding of pseudolymphoma. Immunohistochemical staining of Ki-67 was 39.8% positive in the tumor. Computed tomography revealed no abnormal findings. We diagnosed the tumor as squamous cell carcinoma (SCC) and

treated it with surgical resection². Histopathological examination of the resected specimen showed proliferation of atypical keratinocytes invaded through the dermis with keratinization (Fig. 1G, H). No recurrence has been observed for 3 years.

CLH with erosive lesion healed by itself at first. Then, SCC rapidly grew at the same site. We considered there was an association between CLH and SCC, as SCC developed at the same site of CLH. To our knowledge, this was the first case of SCC developed at the site of CLH. Although chronic injury might cause CLH as well as SCC³, she did not smoke, drink alcohol, or work outside. Therefore, there is a possibility that chronic inflammation for more than 3 years due to CLH caused SCC in our case, although the exact reason why SCC developed at the site of CLH is not clear. There are other possibilities that this case might be merely the coexistence of CLH and SCC, or immune evasion mechanisms are likely to occur before becoming malignant⁴, but SCC occurred at the site where CLH existed. Therefore, dermatologists should keep in mind that SCC could develop at the site of CLH and should follow up patients with CLH.

CONFLICTS OF INTEREST

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