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A Case Report of Spitzoid Melanoma in a Patient with Breast Cancer

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Dear Editor:

A previous study showed increased risks of cutaneous melanoma in patients with breast cancer in Western populations[1]. Asians have a lower incidence of melanoma, with the most common type being acral lentiginous melanoma[2].

A 42-year-old female presented with a 1-cm sized brown nodule and a separate, small brown macule on her right temple. The lesion had appeared the preceding month (Fig. 1A). A year prior, she had been diagnosed with cancer of the right breast. Punch biopsy of skin lesion revealed epithelioid melanocytes with pleomorphic, hyperchromatic nuclei that formed a tumor mass with coarse melanin granules (Fig. 2A, B). A high mitotic rate (3/mm^2) was also observed in the deep dermis (Fig. 2C). The satellite lesion showed an atypical epithelioid melanocytes (Fig. 2D). Immunohistochemical staining of these cells revealed MelanA, Human Melanoma Black-45, and Ki-67 positivity (Fig. 2E~G). Immunohistochemistry for p16 was negative (Fig. 2H). The patient was finally diagnosed with cutaneous melanoma of the spitzoid subtype. After diagnosis, she was transferred to the department of plastic surgery to undergo wide excision (Fig. 1B, C). On preoperative radiological evaluation including ^{18}F-FDG PET/CT, brain magnetic resonance imaging, chest and abdominal CT, any evidence of metastasis was not observed. After lymphatic mapping, a wide local excision with a 1.5-cm margin and sentinel-node biopsy were performed. Breslow depth was 2.77 mm. Tumor-

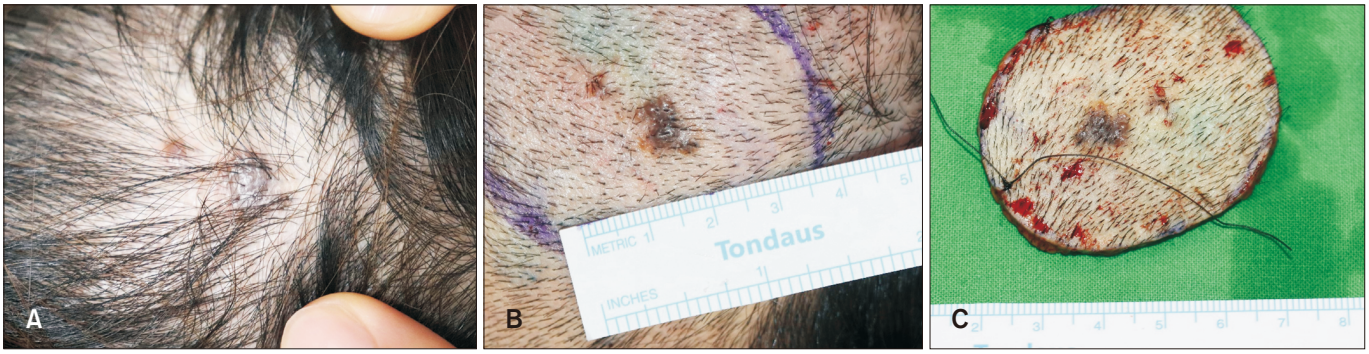

Fig. 1. (A) A dark brown nodular lesion with a brown macule on the patient's right temple. (B) Before excision, (C) wide excision specimen. We received the patient's consent form about publishing all photographic materials.

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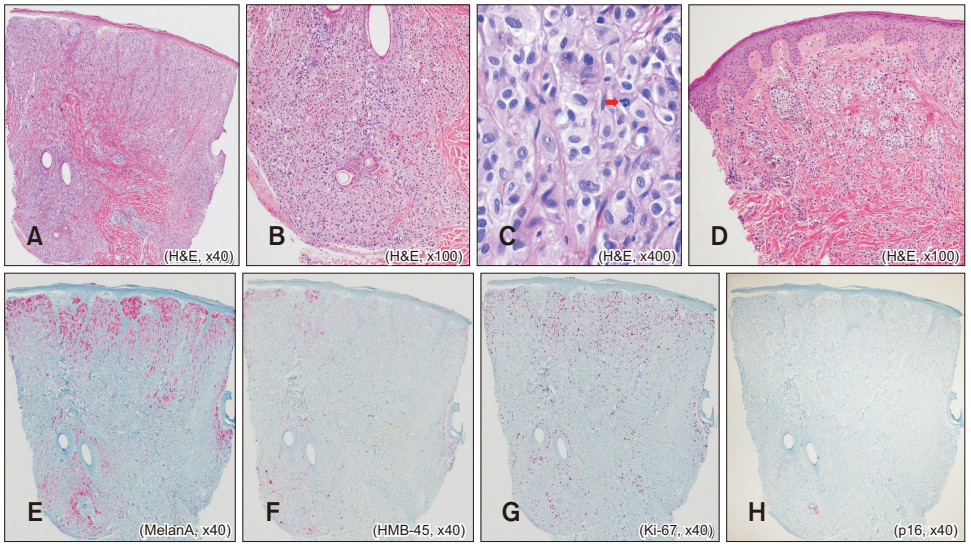

Fig. 2. (A, B) Histologic examination shows an epithelioid-shaped melanocyte, with pleomorphic, hyperchromatic nuclei, forming a tumor mass with coarse melanin granules. A cleft between the nests of melanocytes and adjacent keratinocytes is observed. The epidermis is thin, and Kamino bodies are absent (H&E; A: ×40, B: ×100). (C) The deep dermis also demonstrates a high mitotic rate (arrow). Nested melanocytes within the deep dermis lack maturation (H&E, ×400). (D) A satellite lesion also shows a nested pattern of atypical epithelioid melanocytes (H&E, ×100). (E) The tumor cells are diffusely positive for MelanA (MelanA, ×40) and (F) Human Melanoma Black-45 (HMB-45, ×40). (G) Ki-67 index has been estimated as 50% in the superficial dermis and 20% in the deep dermis (Ki-67, ×40). (H) Negative for p16 (p16, ×40).

infiltrating lymphocytes were observed. Tumor-positive sentinel lymph nodes (right mastoid process area) were confirmed, and radical neck lymph node excision was performed immediately. On pathological staging, stage IIId was confirmed (pT3aN3cM0).

We have tested for *BRAF* V600E and its mutation was detected. Spitzoid melanoma should also be distinguished from nevoid melanoma and epidermotropic metastatic melanoma. Nevoid melanoma usually shows smaller nuclei and less cytoplasm[3]. Epidermotropic metastatic melanoma is usually characterized by the presence of vascular invasion and shows focal epidermal involvement and a wider dermal component which were not seen in the present case[4]. Based on the histological findings, spitzoid melanoma was the most appropriate diagnosis.

With improvements in survival rates of patients with breast cancer, the prevalence of second primary malignancies has increased. One potential explanation for this association is that radiotherapy increases the risk of other cancers. Another possible mechanism for this phenomenon is mutation in cyclin-dependent kinase inhibitor 2A (*CDKN2A*) or breast cancer 2 (*BRCA2*). However, our patient tested negative for *BRCA2* mutation. *CDKN2A* is a tumor suppressor gene that encodes two protein products, namely, p16, a cyclin-dependent kinase inhibitor, and p14[5]. Previous studies have suggested a tumor suppressor role for p16/CDKN2A in melanoma[5]. In our patient, immunohistochemical staining for p16 was negative, and this absence of p16 expression is associated with a loss of a genomic *CDKN2A* copy[5]. Unlike B-RAF and N-RAS, loss of the *CDKN2A* gene has been suggested as a useful tool for the diagnosis of spitzoid melanoma, supporting the hypothesis that this type of cancer is associated with distinct pathways[5].

This case suggests that physicians should be aware of the possibility of melanoma as a second primary cancer in patients with breast cancer.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Hair Regrowth in a Recent-Onset Scarring Alopecia Associated with Kikuchi-Fujimoto Disease

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Dear Editor:

Kikuchi-Fujimoto disease (KFD) is a rare autoimmune disease characterized by cervical lymphadenopathy and fever[1]. It is a self-limiting disease that is more common in Asian populations[1]. Typical cutaneous features include erythematous papules and plaques, and there has been one case of alopecia associated with KFD[2]. Scarring alopecia involves permanent injury to hair follicles and is associated with infection and autoimmune diseases such as lupus erythematous and polymyositis[3]. Since the lesions are refractory to various treatments, early recognition of the disease is helpful in stalling disease progression.

A 44-year-old female presented with an 8 cm pigmented hairless patch on the occiput area with multiple inflammatory nodules that developed 1 month ago (Fig. 1A, B). She experienced mild fever and neutropenia with swollen cervical lymph nodes 4 months ago and was diagnosed with KFD after a lymph node biopsy. Two weeks of oral steroid treatment alone improved tender lymph nodes, leaving a hairless patch. Blood tests were negative for antinuclear antibodies, thyroid dysfunction, and syphilis. Histological examination showed miniaturized hair follicles and a prominently dense infiltration of

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